HUNTINGTON'S DISEASE IN TASMANIA

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CERTIFICATION

This compilation of papers is submitted for the degree of Doctor of Medicine.

The data was collected and the papers written during a five year period from 1986 to 1990, while the author was a member of the academic staff of the Department of Psychiatry, University of Tasmania. For most of this time Professor Ivor Jones was the Head of that Department.

The data was collected by the author and agents acting under his instructions.

The papers were written by the author of this document. Eleven have been published, accepted for publication or presented to publishers for assessment. In two, co-authors have been cited. However, in both, the author of this document was the strategist and senior author. The others were resource personnel with input limited to specific tasks of computing and statistics.

While much of this document has already been published, none of it has been submitted for any other degree at any other institution.

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CONTENTS

GENERAL INTRODUCTION

Huntington's disease (HD) is an inherited, progressive, neuropsychiatric, degenerative disorder which predominantly affects the basal ganglia and cerebral cortex. The onset is usually in adult life, frequently after offspring have been produced.

Others (1,2) had noted the disease earlier in the nineteenth century, but Dr George Huntington, a General Practitioner of Long Island, New York , wrote the definitive description in 1872 (3), at the age of 22 years, one year after his graduation from Colombia University.

> 'I recall it vividly....Driving with my father through a wooded road...we suddenly came upon two women, mother and daughter, both tall, thin, almost cadaverous, both bowing, twisting, grimacing. I stared in wonderment, almost fear.....
>
> ....The hereditary chorea, as I shall call it, is confined to certain and fortunately few families and has been transmitted to them, an heirloom from generations way back in the dim past. It is spoken of by those in whose veins the seeds of the disease are known to exist, with a kind of horror,.....It is attended generally by all the symptoms of common chorea.....
>
> .....There are three marked peculiarities in this disease: 1. its hereditary nature; 2. a tendency to insanity and suicide; 3. its manifesting itself as a grave disease only in adult life.'

Folstein (4) found no convincing descriptions of this disease in the ancient writings. She raises possible explanations: that the first mutation may have occurred in the Middle Ages, and that the human life span may have been too short to reveal the hereditary nature.

Further, she speculated that Huntington's description of the disease received ready acceptance because it arrived at a time when doctors were beginning to appreciate the concept of heredity. But, the brilliance of Huntington's report should also be acknowledged. On this matter Sir William Osler (5) commented, 'there are few instances in the history of medicine in which a disease has been more accurately, more graphically or more briefly described.'

Adams and Victor (6) state the disease can be distinguished by the triad of dominant inheritance, choreoathetosis and dementia. Stanley Fahn (7) also describes a diagnostic triad, but he omits the dominant inheritance feature and adds 'emotional disturbances' to chorea and dementia. There are dangers in attempting to simplify the symptoms of the disease. The 'emotional disturbances' overlooked in the first mentioned triad are frequently the most difficult symptoms. Further, Mattsson (8) and Hayden (9) found that patients first presented with psychiatric symptoms alone, more often than with neurological symptoms alone. In the early stages irritability and impulsivity are evidence of personality change. Depression of both reactive and organic origin occur, and suicide is not uncommon. A schizophreniform psychosis may be the presenting complaint and delusions and hallucinations may continue to be troublesome. In addition to chorea, Folstein (4) has described other involuntary movements (motor restlessness, dystonia, tremor and myoclonic jerks) and abnormalities of voluntary movements (eye movements, fine motor coordination, speech, dysphagia and gait).

The brain displays a symmetrical and severe atrophy involving predominantly the frontal and temporal lobes. Atrophy may also extend into the parietal and occipital lobes. The lateral ventricles

are enlarged, particularly the frontal horns. The caudate is severely atrophied and may be concave where it projects to the surface of the lateral ventricle. Histologically there is diffuse neuronal loss in the cerebral cortex, basal ganglia, thalamus, inferior olives, and anterior horn cells of the spinal cord (10,11).

The biochemical events are unclear. The most consistent finding is a reduction in brain GABA content (12). The literature contains conflicting reports of changes is the various neurotransmitter systems. Borison et al (13) suggested this may be explained by the fact that HD is a progressive disorder, and analyses are being performed on brains obtained at different stages of illness.

The impact of this disease on other members of the affected person's family is profound. Hans and Gilmore (14) stated that while some children do not inherit the gene, none 'escape the disease'. Folstein (15) found there was a high frequency of conduct disorder in the children of affected individuals and attributed this to the social environment of the home rather than the early expression of the gene. Hans and Koppen (16) have drawn attention to the impact of the disease on the unaffected spouse.

The difficulties of young people living 'at risk' are now receiving attention (17). In Australia discussion groups and support activities are organised in every state by the local branch of the Australian Huntington's Disease Association.

A most important advance in the field was the discovery (18) of a marker linked to the Huntington gene on the short arm of

chromosome 4. Subsequently, the ethical and policy issues have been discussed (19,20,21), and adult presymptomatic (22) and antenatal foetus testing has become available (23).

Major difficulties arise from the fact that the G8 probe attaches close to the Huntington gene and allows only a linkage test. This means that blood (DNA) is required from various generations and that, at best, the results are less than 100% accurate. This situation causes practical and ethical problems. The widely held view is that as soon as the abnormal gene can be precisely identified, it will be a simple, rapidly achievable task to determine its function and thence an effective treatment. Advice from experts in molecular genetics confirms that the identification of the abnormal gene is probably only two years away, but they caution that a treatment is not an easy consequence, and is unlikely to follow with great rapidity.

This introduction has avoided the issues of prevalence, fertility, ages of onset and death, and other issues, including early diagnosis, which are the subject matter of this thesis.

REFERENCES

1. Elliotson J. St. Vitus Dance. Lancet 1832; 1: 162-165.

2. Lyon J. Chronic hereditary chorea. American Medical Times 1863; 7: 289-290.

3. Huntington G. On chorea. Medical and Surgical Reporter 1872;

317-321.

4. Folstein S. Huntington's disease. Baltimore: The John Hopkins University Press: 1989: 6.

5. Osler W. Historical note on heriditary chorea. Neurographs 1908; 1: 113-116.

6. Adams R, Victor M. Principles of Neurology. New York: McGraw-Hill, 1977: 932.

7. Fahn S. The choreas. In: Wyngaarden J, Smith L. eds. Cecil Textbook of Medicine, 18th ed. Philadelphia: W B Saunders,1988: 2147.

8. Mattesson B. Clinical, genetic and pharmacological studies in Huntington's chorea. UMEA University Medical dissertations 7. UMEA, Sweden. 1974: 21-51.

9. Hayden M. Huntington's chorea in South Africa. PhD thesis, University of Cape Town. 1979.

10. Rosenberg R. Neurogenetics. New York: Raven Press, 1989, 100-102.

11. Martin J. Huntington's disease. Neurology (Cleveland) 1984; 34: 1059-1072.

12. Martin J, Gusella J. Huntington's disease. The New England Journal of Medicine 1986; 315: 1267-1275.

13. Borison R, Hitri A, Diamond B. Biochemical and pharmacological aspects of movement disorders in Huntington's disease. In: Shah N, Donald A. eds. Movement disorders. New York: Plenum.1986: 275-292.

14. Hans M, Gilmore T. Social aspects of Huntington's chorea. British Journal of Psychiatry 1968; 114: 92-98.

15. Folstein S, Franz M, Jensen B, Chase G, Folstein M. Conduct disorder and affective disorder among the offspring of patients with Huntington's Disease. Psychological Medicine 1983; 13: 45-52.

16. Hans M, Koeppen A. Huntington's disease. The Journal of Nervous and Mental Disease 1980; 168: 209-214.

17. Phillips D. Huntington's Disease. Sydney: A.H.D.A. 1984.

18. Gusella J, Wexler N, Conneally P. A polymorphic DNA marker genetically linked to Huntington's disease. Nature 1983; 306:234-238.

19. Turner D, Haan E, Jacka E, Kalucy R, Burns R, Willoughby J, Crabb R. Prenatal and adult presymptomatic testing for Huntington's disease. Medical Journal of Australia 1988 ; 148: 567-573.

20. Shaw M. Testing for the Huntington gene: a right to know and a right not to know, or a duty to know. American Journal of Medical Genetics 1987; 26: 243-246.

21. World Federation of Neurology: Research Committee Research Group on Huntington's chorea. Ethical issues policy statement on Huntington's disease molecular genetics predictive test. Journal of the Neurological Sciences 1989; 94: 327-332.

22. Meissen G, Myers R, Mastromauro C. et al. Predictive testing for Huntington's disease with use of a linked DNA marker. New England Journal of Medicine 1988; 318: 535-542.

23. Quarrell O, Meredith A, Tyler A, Youngman S, Upadhyaya M, Harper P. Exclusion testing for Huntington's disease in pregnancy with a closely linked DNA marker. Lancet 1987; ii: 1281-1283.

THESIS INTRODUCTION

This compilation of papers is the product of five years of research and clinical involvement in Huntington's Disease (HD) in Tasmania.

In 1949, Dr Charles Brothers published some details of a large, Tasmanian HD pedigree (1). Others performed calculations on his figures (2,3) and stated that the region had one of the highest per capita rates in the world. Later, the Department of Psychiatry, of the University of Melbourne, took an interest in Tasmanian HD families. However, no further information was published until that which appears herein.

In 1986, the author commenced research and clinical practice in HD.

First, the functions of HD Registers (HDRs) were examined. This led to the establishment and maintained of an HDR. This, in turn called for the establishment of an HD Advisory Committee. These events have been described.

Pure research was subsequently conducted on the prevalence of HD in Tasmania, the age of onset, the age of death and the duration of the disease, the fertility of affected individuals and their unaffected siblings, and sundry features of the total HD population and of the large HD family earlier described by Dr Brothers.

The above findings were used as authorative data in a review of the social justice issues of HD in Tasmania. This review favourably influenced government policy and funding.

Concurrently, a study of early detection techniques using neuropsychological testing, neurological examination and Quantitative (Q)EEG has been running for two years. Final data is not yet available and thus results can not be presented, but a single case study of an individual examined with QEEG before and after the clinical onset of HD is described.

The appendix contains two sets of data. The first is details of the large Tasmanian which at January 1, 1990, extended over 43 meters of paper. The second is complete details of the neurophysiological results of the single case mentioned above.

REFERENCES

1. Brothers C. The history and incidence of Huntington's chorea in Tasmania. Proceedings of the Royal Australasian College of Physicians 1949; 4: 48-50.
2. Myrianthopoulos N. Huntington's chorea. Journal of Medical Genetics 1966; 3: 298-313.
3. Conneally P. Huntington's disease: genetics and epidemiology. American Journal of Human Genetics 1984; 36: 506-526.

CHAPTER 1.

THE TASMANIAN HD REGISTER

The section of this chapter dealing with the functions of a register have been published in the Australian and New Zealand Journal of Psychiatry 1989; 23: 161-162.

a. PREAMBLE

In 1949 Dr. Charles Brothers, the Director of Mental Hygiene in Tasmania, published an internationally acknowledged account of HD in the state (1). He reported on five generations of a single pedigree.

Subsequently, episodes of data collection were supervised by Dr. John Weatherly, Psychiatrist Superintendent, Royal Derwent Hospital, Tasmania, Dr. Eric Cunningham Dax, Co-ordinator of Community Services, Mental Health Services Commission, Tasmania, Dr. Edmond Chiu and Mrs. Betty Telscher, Department of Psychiatry, University of Melbourne, and Dr. Keith Millingen, Reader, Department of Medicine, University of Tasmania.

Unfortunately, these earlier efforts had been largely wasted. Records were frequently either lost, unintelligible (by dint of faded ink or idiosyncratic inscription), or contradictory. For example, even the names of the individuals on the pedigree illustrated in Dr. Brothers paper were missing, and it was believed some of the relationships were inaccurate.

The most useful records were those offered by the University of Melbourne group. But these too were incomplete, in part inaccurate, and limited to certain branches of certain families, in certain localities of the state.

As a useful Huntington's disease register (HDR) did not exist, and establishment of such would require the expendature of effort, time and money, a study of the functions of HDRs was conducted. The findings have been published (2) and are presented below.

b. THE FUNCTIONS OF AN HD REGISTER

1. Diagnostic Activities

(a) In Clinical Medicine.

One of the three diagnostic criteria of HD is a positive family history (3,4). Thus, the Register is a source of clinically important details, especially in isolated or difficult cases (5).

(b) In Laboratory Medicine

Recent developments in molecular biology techniques have made pre-symptomatic (6,7) and prenatal (8,3) testing possible. As these are linkage tests, DNA (blood) is required from various other family members. An HDR allows identification and location of the full range of possible participants. (9). Additionally, the HDR is a convenient means of keeping a record of those specimens which have already been obtained and stored, either in anticipation of, or response to earlier, requests.

2. Clinical Care Role

(a) Clinical Features

Some families have a particular age range of onset and death, and characteristic symptom pattern (1,10).

(b) Treatment

When a significant advance in treatment is achieved, an HDR will enable workers to identify and locate those who will benefit.

3. **Service Monitoring**

An HDR enables the determination of a regional prevalence (11) and then, the appropriate distribution of services. It will also enable assessment of the efficacy of genetic counseling programmes (12)

4. **Research**

An HDR is a collection of information which invites epidemiological research. It may also form a basis for experimental research.

5. **Preventive Activities**

(a) Formal Genetic Counselling

An accurate pedigree is essential for genetic counselling (13).

(b) Informal Genetic Counselling

The process of keeping an HDR reduces the prevalence of HD. It has been found that family members do not understand the true hereditary nature of the disorder and welcome advice (5). It has been found that 85% of relatives at high risk of having a child with a serious genetic disorder are unaware of the risks (14). In the process of keeping a HDR, ample opportunities present for informal genetic counselling and this prevents disease (15).

6. Cost Saving

Through the prevention of disease, HDR's will save much more money than they cost to establish and maintain (12,16,17).

7. Educational Function

Molecular biology is an unprecedented advance in the diagnosis and prevention of a wide range of medical conditions (18,19). From the clinician's point of view the genetics of HD is relative simple. The HD marker is "arguably the most important application of medical genetics so far" (20) and the HDR is an excellent model from which to learn.

8. Morale Boosting Function

(This additional function was not apparent from the review of the literature. It is a useful function learned of through practical experience.)

An HDR has a morale boosting function. Contributing information to a HDR gives the lay community the opportunity for constructive activity. Further, the existence of a HDR has been observed to bring people together and encourage valuable mutual support.

c. ESTABLISHMENT OF THE CURRENT REGISTER

There had been episodic pressure by medical practitioners (21), members of HD families and the Australian Huntington Disease Association, Tasmanian Branch (AHDATas) (22), on the state government and government agencies, for assistance.

Consequently, the Mental Health Services Commission (MHSC) requested the above study and in light of the findings, decided in favour of the establishment and maintenance of an HDR.

The MHSC contracted the author to supervise the task and provided an equipment fund and a half-time social worker. The AHDAT as also provided two amounts of funds over subsequent years.

d. METHOD

i. Recording

As mentioned, many of the records made in the past were found to be useless due to loss, illegibility and contradiction. With this example in mind, the decision was made to use an established coding system (23). Two paper copies were drawn. The master copy was held in the author's office and the working copy was used in the field. New data was first entered on the working copy and later, after consideration, entered on the master copy. This arrangement had advantages: a review mechanism in the two stage process of data acceptance, and the security of having (at almost all times) copies of the data in two separate geographical locations.

The disadvantages of paper copies of pedegrees include that they are physically large and time consuming to access. These disadvantages considerably reduces the usefulness of an HDR to clinical practioners.

Accordingly, the decision was made to establish, in addition to

the paper copies, a computerised system. After assessment of the alternative programmes, "The Huntington's Chorea Clinical Register System V2.2" (HCCRS), developed by the Institute of Medical Genetics, University of Wales College of Medicine, Cardiff, was found to be the most suitable for the Tasmanian situation. Subsequently, a programme for the production of line drawings of pedigrees ("PLOT 2000") was obtained from the same institution.

An IBM compatible PC was purchased, dedicated to HD and housed in the office of the author.

Initially, data was entered by the author. Later, research assistants were employed in this way. Finally, when the data base was accepted as being clinical useful, a part-time MHSC computer operator was made available.

ii. Data Acquisition

An exhaustive search for data was conducted. This involved the following individuals, institutions and methods:

i. All available previous register material was examined.

ii. Contact was made with members of all known families.

iii. The cooperation of the AHDATas was sought and obtained.

iv. Records were obtained by the author from:

1. Royal Hobart Hospital
2. Royal Derwent Hospital, New Norfolk
3. Launceston General Hospital
4. Lindsay Miller Clinic, Launceston

5. North West General Hospital, Burnie
6. Eskleigh Nursing Home, Perth (Tasmania)
7. St John's Park, Hobart
8. Aralee Nursing Home, Hobart
9. Strathaven Home For The Aged, Hobart
10. Department of Pathology, University of Tasmania
11. Department of Community Health, Uni. of Tasmania
12. Melrose, MHSC, Tasmania

v. Medical Practitioners with a known interest in the field were contacted

vi. General Practitioners were circulated via the RACGP newsletter

vii. Publicity was obtained by newspaper, television and an international conference held in Launceston

viii. Data was also obtained from Registrar-General's Division, Australian Archives, councils, churches, museums, libraries and graveyards.

e. EFFICIENCY OF ELECTRONIC REGISTER

The HCCRS programme proved to be fast and reliable. By this method, the goal of being able to respond to requests for information without delay, was satisfactorily achieved.

There were some 'bugs' in the programme, which episodically required the assistance of a computer systems officer. However, these were correctable and did not reappear.

Some HCCRS options were very slow. For example, the 'Standard Analysis' option, which is a comprehensive statistical analysis of all data on the main file, takes around 12 hours, when the main file contains 4000 individuals. However, these delays were restricted to the statistical options and do not prevent rapid response to requests for information regarding one or a small number of individuals.

'PLOT 2000' has been of limited use in providing diagnostically useful information. However, it has been very useful when requests have been made regarding the possibility of obtaining blood from relatives for presymptomatic and antenatal testing.

f. UTILASATION OF THE REGISTER

The HDR has proved to be a useful clinical service. During 1987, 1988 and 1989 there were 17, 23 and 29 requests for information, respectively. The increasing number of requests reflect an increasing awareness of the existence of the Tasmanian HDR.

Requests for information came from two sources: Tasmania and mainland Australia.

Requests from within Tasmania came predominantly from treating medical practioners who wanted information to support or exclude a diagnosis. On fewer occasion it was sought by young people (through their general practioners) to assist with family planning decisions.

Requests from mainland Australia have come from medical practioners seeking diagnostic assistance, and from other HDRs. The

keepers of other HDRs around Australia, who have individuals of Tasmanian origin in their state, have been keen to complete their existing information. There have also been requests from mainland workers seeking information to be used in presymptomatic and antenatal testing programmes. The question in these cases being whether their clients had close relatives living in Tasmania who could be approached for DNA samples.

The requests from other HDRs have become less frequent. However, the total number of requests are unlikely to decline, particularly if genetic testing becomes available locally.

As far as is known, there was no attempt to obtain information from the HDR by any individual, or organisation, for purposes other than diagnosis, family planning and genetic testing.

g. RULES FOR ACCESS TO THE REGISTER

A danger in maintaining an HDR is that the information could be accessed and used to the disadvantage of listed individuals. For example, insurance and superannuation have been refused to symptom free individuals who were known to be members of HD families. This is of great concern in Tasmania because of the small population and high prevalence of the disease. On this account, there was reluctance to be listed on the HDR by some 'at risk' individuals.

The MHSC had created a Huntington's Disease Advisory Committee (HDAC) to address issues in the area of HD. This committee is the topic of the next chapter. However, for the sake of continuity, the rules which it developed for the maintenance of and access to, the

computerised HDR, are presented here:

HUNTINGTON'S DISEASE ADVISORY COMMITTEE

Rules for Maintenance of and Access to the Computer Register

of members of Huntington's Disease Families

1. General Principles

1.1 The Huntington's Disease Advisory Committee, a body established by the Mental Health Services Commission, recommends the establishment of a computer register of members of families affected by Huntington's disease for the purpose of data collection and bona fide research.

1.2 All information relating to a person on the register is confidential and may be dealt with only in accordance with these procedures.

1.3 When data is provided by any person for the register that person shall be given a copy of these Rules.

1.4 The Chairman for the time being of the Huntington's Disease Advisory Committee shall be such person as is appointed by the Mental Health Services Commission.

2. Maintenance of Register

2.1 The Register will be maintained on three (3) separate sets of computer discs at the Faculty of Medicine, University of Tasmania.

2.2 The discs must be kept in secure and separate places and one(1) at least shall be kept in the safe of the Faculty of Medicine.

2.3 One copy of the Register shall be kept in a fireproof cabinet.

3. Access to the Register

3.1 The following persons may have access to the data in the register,

(a) Chairman of the Huntington's Disease Advisory Committee,

(b) Any person approved by a meeting of the members of the Huntington's Disease Advisory Committee, and,

(c) Any bona fide researcher provided that the prior approval of the Huntington's Disease Advisory Committee has been obtained and that any conditions as to the use of data laid down by the Huntington's Disease Advisory Committee are scrupulously observed.

3.2 The persons whose data appears on the register may

have access to their own personal data and this personal data may be provided by the Chairman of the Huntington's Disease Advisory Committee.

3.3 With the exception of those persons in 3.1 and 3.2, data on a person on the register shall not be given to any other person except in accordance with paragraph 4 of these Rules set out below.

4. Request for Information

4.1 Formal Requests for Information

4.1.1 No request for information from the Register will be considered unless the request is from a registered Medical Practitioner and such a request must be in writing.

4.1.2 Any information provided by the Chairman of the Huntington's Diseased Advisory Committee must be in writing and given with the written consent of the person or persons concerned.

4.2 Urgent Requests for Information

4.2.1. The Chairman of the Huntington's Disease Advisory Committee may answer requests for information by telephone where the Chairman considers it expedient so to do.

4.2.2. In all cases of telephone requests for information the Chairman must,

(a) obtain the telephone number of the person requesting the information and return the call by way of verification,

(b) enter a diary note about the date, time, duration and nature of the request for information, and

(c) make a report to the next scheduled meeting of the Huntington's Disease Advisory Committee for consideration.

4.3 Annual Report on Requests for Information

The Chairman of the Huntington's Disease Advisory Committee shall annually present the Huntington's Disease Advisory Committee by a report on the numbers and types (normal or urgent) of requests for information received during the year.

5. Problems in relation to the Register

5.1 If any problem arises in relation to the maintenance of the register, data thereon or access thereto, the Chairman shall in the first instance take such steps as are necessary to resolve the problem.

5.2 Any action taken by the Chairman under 5.1 must be reported to the next scheduled meeting of the Huntington's Disease Advisory Committee for consideration.

5.3 If the Chairman cannot resolve the problem, the problem shall be referred to the Huntington's Disease Advisory Committee for consideration.

5.4 In all cases where the problems concerns a person on the register, this person shall be consulted and may attend any meeting of the Huntington's Disease Advisory Committee which considers the problem.

h. REFERENCES

1. Brothers C. The history and incidence of Huntington's chorea in Tasmania. Proceedings of the Royal Australasian College of Physicians 1949; 4: 48-50.

2. Pridmore S. Huntington's disease registers. Australian and New Zealand Journal of Psychiatry1989, 23:161-162.

3. Hayden M. Huntington's chorea. New York: Springer-Verlag, 1981.

4. Adams R, Victor M. Principles of Neurology. New York: McGraw Hill,1977

5. Pleydell M. Huntington's chorea in Northamptomshire. British Medical Journal 1954; 11: 1122 - 1128.

6. Gusella J, Wexler N, Conneally P. A polymorphic DNA marker genetically linked to Huntington's disease. Nature 1983; 306:234-8.

7. Meissen G, Myers R, Mastromauro C. et al. Predictive testing for Huntington's disease with use of a linked DNA marker. New England Journal of Medicine 1988; 318: 535-42.

8. Quarrell O, Meredith A, Tyler A, Youngman S, Upadhyaya M, Harper P. Exclusion testing for Huntington's disease in pregnancy with a closely linked DNA marker. Lancet 1987; ii: 1281-1283.

9. Harper P, Sarfarazi M. Genetic prediction and family structure in

Huntington's chorea. British Medical Journal 1985; 290 :1929-31.

10. Folstein S, Phillips J, Myers D, et al. Huntington's Disease: two families with differing clinical features show linkage of the G8 probe. Science 1985; 229:776-779.

11. Reed T, Chandler J. Huntington's chorea in Michigan. American Journal of Human Genetics 1958; 10: 201-225.

12. Emery A, Brough C, Crawford M, et al. A report on genetic registers. Journal of Medical Genetics 1978; 15: 435-42.

13. Emery A, Smith C. Ascertainment and prevention of genetic disease. British Medical Journal 1970; 3: 636-7.

14. Harper P, Walker D, Tyler A, et al. Huntington's chorea. The Lancet 1979; 2:346-349.

15. Harper P, Tyler A, Smith S, Jones P. Decline in the predicted incidence of Huntington's chorea associated with systematic genetic counseling and family support. Lancet 1981; ii: 411-413.

16. Carter C, Evans K, Baraitser M. Effect of genetic counseling on the prevalence of Huntington's chorea. British Medical Journal 1983; 286: 281-283.

17. Staples A. The cost of Huntington's disease in South Australia. Draft Report to the Ad Hoc Genetic Services Committee, South Australian Health Commission, February 1987.

18. Weatherall D. The new genetics and clinical practice. 2nd ed. Oxford: Oxford University Press, 1985.

19. Martin J. Molecular Genetics: applications to the clinical neurosciences. Science 1987; 238:765-772.

20. Harper P. A genetic marker for Huntington's chorea. British Medical Journal 1983; 287: 765-72.

21. Letter from Dr E C Dax to Medical Commissioner, Dr P Eisen, 1981.

22. Minutes of the Australian Huntington's Disease Association, Tasmanian Branch, 1976-85.

23. Yesley G. Methods of coding and filing family records. In: Stanbury J, Wyngaarden J, Fredrickson D, eds. The metabolic basis of inherited disease. New York: McGraw-Hill, 1966; 1415-24.

CHAPTER 2.

THE HD ADVISORY COMMITTEE

a. PREAMBLE

By early 1987 it became clear that the HDR, storage of blood and the possibility of genetic testing were raising new issues which required discussion and in-put from a wide range of interested individuals and groups.

At that time the author was Clinical Commissioner of the Mental Health Services Commission (MHSC) and was influential in the establishment of the Huntington's Disease Advisory Committee (HDAC).

b. ESTABLISHMENT

The HDAC was established as a statutory body under Section 8 of the Mental Health Services Act,1967. This Section allows the appointment of committees to advise or assist the Commission on specific matters.

To achieve depth of interest and experience the HDAC was composed of the following:

1. The Clinical Commissioner (Chairman) or a nominee.
2. A representative of the Australian Huntington's Disease Association, Tasmanian Branch (AHDATas).
3. A health professional from the MHSC staff. (This

position ordinarily to be occupied by the relevant MHSC social worker.)

4. A representative of a Department of the Faculty of Medicine, University of Tasmania, other than the Department of Psychiatry.
5. A member of the legal profession.
6. Such others as may be determined by the MHSC. (As the elected representative of the AHDATas was not an HD family member, the HDAC requested the MHSC use this provision to appoint an 'at risk' individual.)

c. TERMS OF REFERENCE

The MHSC determined the terms of reference of the HDAC to be to advise and assist 'on matters generally relating to Huntington's Disease and specifically the ethical and practical issues relating to-

1. maintenance of an HD Register;
2. access to that Register;
3. predictive testing;
4. appropriate research; and
5. deployment of resources'

The term of office was two years (Manton, 1987).

d. DELIBERATIONS AND DECISIONS

The HDAC met monthly for four months and bimonthly thereafter. At the end of two years the MHSC requested the members to stand for another term.

First, the deliberations and decisions of the HDAC will be reported according to the terms of reference set out above:

1. Maintenance of an HDR

   The HDAC considered the fledgling HDR to be a useful and worthy of continuing support.

2. Access to the HDR

   This topic received lengthy and repeated discussion. There was unanimous support for measures to preserve confidentiality. A set of Rules of Access were developed and widely circulated. They have been detailed in an earlier chapter. The HDAC accepted the view expressed earlier by the AHDATas, that a dedicated PC be used for the purpose rather than a less secure main-frame computer.

   At every meeting the author reported (without explicit detail) the number and nature of requests received.

   The HDAC inspected the HDR security mechanisms.

3. Predictive Testing

   The HDAC developed the view that, given the limited support services available in the state, antenatal testing is preferable to presymptomatic testing.

   The HDAC recommended the storage of DNA against the day the testing became available. It took an interest in and inspected the storage

facilities made available at the designated laboratory.

At the time of writing the HDAC had begun to to formulate detailed recommendations on establishing antenatal testing in the state which are to be presented to the government.

4. Appropriate Research

The HDAC initiated one research project. This was mail survey of the desire for genetic testing among HD family members. This was conducted by the AHDATas.

It reviewed and suggested minor refinements in the procedures to protect confidentiality of a number of studies on HD.

5. Deployment of Resources

The HDAC made suggestions to the MHSC regarding the deployment of human resources, recommending an additional half-time social worker be made available for HD services.

The MHSC accepted this recommendation in principle but was unable to accede. (Eventually, in mid-1990, due to pressure other than that applied by the HDAC, which will be detailed later, the services of a half-time social worker were acquired.)

It suggested a part-time computer operator be made available and this came to pass.

It supported and passed on to the MHSC, the suggestion of the AHDATas that an HD outpatient

clinic be established at the Launceston General Hospital.

It drew to the attention of the MHSC, the need for the establishment of genetic counseling and and support prior to the commencement of genetic testing.

Finally, the HDAC, spent time on functions best described as additional to the foundation terms of reference. As stated, it was established to advise and assist the MHSC. In practice it also reassured and advised the HD families. It reassured them that the authorities were aware of and interested in their problems. Specific examples of assistance flowing from the HDAC to members of the AHDATas included advice on relatively straightforward legal matters (such as the meaning of power of attorney) which the member of the legal profession (Prof Don Chalmers) was able to provide. There were also occasions when the HDAC could represent both the MHSC and the AHDATas, such as when the author wrote to a newspaper to refute inaccurate published material.

e. EVALUATION

The HDAC has been a very useful organisation. It has conveyed informed, considered opinion, on new and challenging issues, to a state authority. In addition it has given valuable service to members of HD families.

The establishment of such a body in other states is recommended.

Having as Chairperson, an individual with a working relationship

with the state authority reduces the sense of impotence which can damage the morale of interested groups. Of course, this may create some difficulties (which are perhaps best construed as stimuli) for the Chairperson. The benefits for the Chairperson include unique insights and an entree to a museum of pathology.

CHAPTER 3.

HD POPULATION SURVEY OF TASMANIA

This chapter is based on data which will be published in the Bulletin of the Human Genetics Society of Australiasia 1990; Volume 3: Number 3.
The data were used by the HDAC in justifying the establishment of the Tasmanian Antenatal Genetic Testing Service.

****

A study was conducted of the total HD population of Tasmania as of January 1st, 1990. The method of data collection is presented in the next chapter which deals in detail with prevalence. The results follow.

a. SUNDRY TOTALS

These totals were calculated using all known generations (past and present) of all known families.

Total number of individuals in the Tasmanian pedigrees	-	2086
Males	-	997
Females	-	989
Individuals Alive	-	1378
Individuals Dead	-	506
Affected Alive	-	56
Affected Dead	-	276
Prevalence	-	12.8/100,000 (living individuals only)

b. DETAILS OF THE FAMILIES

There were 14 HD families in Tasmania. The twelfth is extremely large and is dealt with at length in Chapter 10.

	FAMILY						
	1	2	3	4	5	6	7
TOTAL IN KINDRED	58	13	37	6	38	24	53
NUMBER AFFECTED (DEAD)	3	2	3	1	5	4	2
NUMBER AFFECTED (ALIVE)	1	0	1	0	4	0	0
NUMBER DEAD	8	3	8	1	19	5	12
NUMBER ALIVE	50	0	21	0	15	1	0
NUMBER AT 50% RISK	16	0	4	0	11	1	0
NUMBER AT 25% RISK	21	0	13	0	0	0	0

	FAMILY						
	8	9	10	11	12	13	14
TOTAL IN KINDRED	138	51	42	14	1568	43	4
NUMBER AFFECTED (DEAD)	18	4	1	3	156	3	1
NUMBER AFFECTED (ALIVE)	7	0	0	2	40	1	1
NUMBER DEAD	64	5	4	8	357	11	1
NUMBER ALIVE	73	1	0	6	1179	32	1
NUMBER AT 50% RISK	20	0	0	4	315	11	2
NUMBER AT 25% RISK	27	0	0	0	536	18	0

c. RISK CALCULATIONS

i. Simple Inheritance Risks

TOTAL ALIVE AT 50% RISK- 382

25% RISK- 615

12.5% RISK- 264

TOTAL ALIVE AT <25% RISK- 324

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ii. Age Modified Risks

When the risks are age modified using the Huntington's Chorea Clinical Register System V2.2, the results are as follows:

TOTAL ALIVE AT >= 40% MODIFIED RISK- 280

>= 25% - 579

>= 10% - 836

TOTAL ALIVE AT <=10% MODIFIED RISK - 486

---

iii. Risk and Reproduction

By conservative estimate, 186 Tasmanians have a simple inheritance risk of 50% and are 42 years of age or younger. Thus, there are at least this many people who could, at some time in the future, request antenatal testing services. This number will increase dramatically as almost half will develop the disease, and their children (on average, at least 2 for each newly affected individual) will then be at 50% risk.

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CHAPTER 8.

THE FERTILITY OF HD AFFECTED INDIVIDUALS IN TASMANIA

This chapter is based on a paper accepted for publication in the Australian and New Zealand Journal of Psychiatry

a. ABSTRACT

A study was conducted of the fertility (number of live births) of Huntington's disease (HD) affected individuals compared to that of the general population (using data from the 1986 Census of Population and Housing). HD affected individuals were found to be at least as fertile as members of the general population. This finding supports earlier studies. The implication for consideration is that once individuals at 50% risk have achieved their desired family size, it is appropriate to offer reversible sterilization.

b. INTRODUCTION

The aim of this paper is to determine the fertility (number of live births) of the affected members of the Huntington's disease (HD) families of Tasmania, compared to that of the general population of the state. This comparison is called the relative frequency (Reed and Neel,1959). Accurate relative fertility data are required for scientific and planning purposes.

Tasmania was identified as a region of high prevalence of HD when Brothers (1) published details of a single, large, resident pedigree. A recent study (2) found a prevalence in this state of 12.1/100,000. This is twice the average occidental prevalence. Tasmania is an island with a stable, relatively small population, which is readily accessed. With a sophisticated medical infrastructure, it is well suited to population health surveys (3).

c. LITERATURE REVIEW

Five studies have compared the fertility of affected individuals with that of the general population (4,5,6,7,8). Four (5,6,7,8) give a relative fertility of affected individuals (AI) over general population (GP) of unity or above. See Table 1.

Reed and Neel (4), wrote an account of HD in Michigan. They used general population data from the US census of 1941. They found a relative fertility (AI/GP) of 0.9. While elaborate, this was less than a total population survey. Surprisingly, they found that affected individuals produced about 2.4 children, considerably less than the 4.8 children of an earlier era (9) and the 5.35 children which can be calculated from the smaller study of a later era (10).

Wallace and Parker (5), working in Queensland, Australia, then found affected individuals produced the same number of children as did the general, non-aboriginal population, and calculated a relative fertility (AI/GP) of unity.

Shokeir (6), working in the Canadian Prairies, Marx (7),

working in Minnesota, and Stevens (8), working in England, found relative frequencies (AI/GP) of 1.1, 1.2, and 1.4, respectively. Stevens complained that the Registrar-General had published two sets of data from the 1961 census of England and Wales. From these he derived two versions of the mean fertility of the general population. When his higher estimate (2.13 children) is used, the relative fertility (AI/GP) remains above unity.

RELATIVE FERTILITY AFFECTED INDIVIDUALS/GENERAL POPULATION (AI/GP)	R+N*	M	W+P	St**	Sh
Number of Children to Affected Individuals	2.37	2.97	3.19	2.36	3.78
Number of Children to General Population	2.58	2.57	3.19	1.73	3.31
Relative Fertility (AI/GP)	0.9	1.2	1.0	1.4	1.1

R+N : Reed and Neel (1959)

* : reworked by Marx (1973)

M : Marx (1973)

W+P : Wallace and Parker (1973)

St : Stevens (1976)

** : using one of two estimates of GP fertility

Sh : Shokeir (1975)

Table 1: Fertility of affected individuals relative to the general population.

d. METHOD

A field survey of the Tasmanian HD families was conducted. Components of the survey included:

1. Examination of all previously compiled, unpublished pedigrees
2. Co-operation with the Australian Huntington's Disease Association, Tasmanian Branch
3. Provision of a Research Social Worker by the Mental Health Services Commission, Tasmania
4. Television and newspaper publicity
5. Circulation of General Practitioners via the RACGP newsletter
6. Collection of data from family members, medical practitioners , hospitals, nursing homes, Registrar General's Division, Australian Archives, libraries, museums, councils, churches and graveyards

The data collected were the date of birth, disease status and blood relationships of the individuals of known HD families. This was entered on a Personal Computer using Huntington's Chorea Clinical Register System V2.2, and manipulated using DBaseIII and Statgraphics programmes. Fertility was determined by counting all the live births by a an individual.

The general population raw data were taken from the Australian Bureau of Statistics 1986 Census of Population and Housing. It was the number of children ever born to the women of Tasmania who were alive in 1986. It was available in sub-divided form, on the basis of the age of the women. The HD groups were therefore selected for individuals alive in 1986, whose age and number of children were known. Males were included as proxies for their partners, and were assumed to be of the same age group. (Males

as proxies has been discussed in the previous chapter and the two relevant published papers.) The general population data was adjusted to meet the age distribution of the affected individuals.

e. RESULTS

Data were available from 26 affected individuals living in Tasmania on July 1, 1986. They produced an average of 2.96 children. The group representing the general population produced an average of 2.85 children. This gives a relative frequency (AI/GP) of 1.04. See Table 2.

RELATIVE FERTILITY

AFFECTED INDIVIDUALS/GENERAL POPULATION (AI/GP)

	No. Individuals	No. Children	Average No. Children	AI/GP
Total Tasmanian HD Population	26	77	2.96	1.04
General Population	26	74.41	2.85	

Table 2. Fertility of the affected individuals of the Tasmanian HD population and the Brothers' family, and the general population.

f. DISCUSSION

This method involves the counting of offspring of living people who have at least achieved the reproductive years. A source of error is that some asymptomatic heterozygotes will not have been counted in the HD group. However, as affected individuals have equal or greater fertility, leaving as yet undiagnosed individuals in the general population group would only serve to artificially elevate the fertility of that group at the expense of the HD group.

The finding of a relative fertility of 1.04 indicates that the Tasmanian HD affected population is as fertile or more fertile (in terms of producing more offspring) than the general population. This is in keeping with the the most recent overseas studies (5,6,7,8).

This is especially remarkable given two facts. First, that to be included in the HD affected group, individuals must have had symptoms, and these symptoms are disabling. Second, that the unaffected siblings of HD families have been found to have a relative frequency of unity (5,7) or below (4,6,8).

Thompson and Thompson (11) have observed that natural selection against HD is not strong since its late onset age means that it does not greatly impair the biological fitness of the heterozygote. In fact, there is evidence to suggest that some feature of the disease increases fertility (when measured by the number of children born). The implication for consideration is that once individuals at 50% risk have achieved their desired family size, it is appropriate to offer reversible sterilization.

g. REFERENCES

1. Brothers C. The history and incidence of Huntington's chorea in Tasmania. Proceedings of the Royal Australasian College of Physicians 1949; 4: 48-50.

2. Pridmore S. The prevalence of Huntington's disease in Tasmania. Medical Journal of Australia 1990, in press.

3. Dwyer T. The future for epidemiology in Tasmania. In: King H, ed. Epidemiology in Tasmania. Canberra: Brolga Press, 1987:259-267.

4. Reed T, Neel J. Huntington's chorea in Michigan. American Journal of Human Genetics 1959; Vol 11: 107-36.

5. Wallace D, Parker N. Huntington's chorea in Queensland: the most recent story. Advances in Neurology 1973; 1:223-36.

6. Shokeir M. Investigation on Huntington's disease in the Canadian Praries. Clinical Genetics 1975; 7: 349-53.

7. Marx R. Huntington's chorea in Minnesota. In: Barbeau A, Chase T, Paulson G, eds. Huntington's chorea1872-1972. New York: Raven Press, 1973: 237-49.

8. Stevens D. Huntington's chorea: a demographic, genetic and clinical study. MD thesis 1976, University of London, 1-338.

9. Bell J. Huntington's chorea. In: Fisher R, ed. The treasury of human inheritance, vol IV/1. London: Cambridge University Press, 1934: 1-77.

10. Lyon R. Huntington's chorea in the Moray Firth area. British Medical Journal 1962; i: 1301-6.

11. Thompson J, Thompson M. Genetics in medicine. 4th ed. Philadelphia: W B Saunders Company. 1986: 75.

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CHAPTER 12

THE TASMANIAN EARLY DETECTION STUDY

The case study in this chapter has been submitted to

Biological Psychiatry

a. INTRODUCTION

Over the last five years, many people 'at risk' for HD have come to the author asking to be examined for any signs of the disease. They have wanted to know so that they could plan the next phase of their lives, reckoning that if the disease is present, they have have a couple of years to make sensible, definitive arrangements, and no time to commence new, long-term initiatives. This experience aroused interest in methods of early detection.

A distinction is drawn between early detection and presymptomatic testing. Presymptomatic testing clarifies whether or not the disease will develop, at some time in the individual's life (1). It is a 'bigger question', it can only be asked once, and if answered in the affirmative, it calls for major personal adjustment. The evidence shows that there are many 'at risk' individuals who do not want this question applied to themselves. For example, one of the investigators who discovered the G8 probe (2) is 'at risk', but has not taken the test. While

some surveys (3) have found the majority of 'at risk' individuals declare a wish for presyptomatic testing, and some HD clinics have found increasing interest (4), others have encountered initial enthusiasm, followed by reluctance (5).

Early diagnosis is an additional service and not an alternative to presymptomatic testing. It has value irrespective of whether or not the presymptomatic test has been taken. The disease has a variable age of onset (6) and individuals who is aware that they carry the abnormal gene will still, in many cases, want advanced warning of the commencement and staging of the disease process.

Neuropsychological testing holds promise as a method of early detection (7-13). In a recent study using neuropsychological and genetic testing (13), five of seven otherwise asymptomatic individuals who were found to have inherited the abnormal gene, showed abnormalities in visuospatial and frontal lobe function. The age of onset of their affected parents was at least twelve years older than the test subjects at the time of the reported testing. Taken together, these studies indicate that neurophysiological impairment may precede other onset signs and symptoms by a number of years.

Positron emission tomography (PET) has demonstrated impairment of caudate glucose utilization in some subjects who appear, on genetic testing, to have inherited the abnormal gene (14). As this method allows visualisation of the metabolic function of key structures, it can be expected to be useful in early detection. The disadvantage of PET is cost. It is not yet available in Australia. Even when it becomes so, the expense of each procedure and the inconvenience of traveling form distant regions, will reduce its usefulness.

CT bicaudate ratio studies of established cases have a sensitivity of only 77% and a specificity of 92% (15). Thus, this technique is of limited value in the early detection of HD.

Traditional electroencephalographic studies in established disease have been disappointing. Early reports (16) described a loss of alpha rhythm and replacement by low voltage slow activity. However, only one third of patients display these features (17,18) and false negatives have been reported in 70% of cases (19, 20). Further, the EEG abnormalities do not reflect the clinical severity of the disease (21) and the follow-up report of a much publicised attempt to predict the disease using EEG (22) did not appear.

Computerization has made possible a range of relatively inexpensive and convenient methods of quantifying and displaying EEG data. Such methods have been applied to the study of dementia (23,24) and may have a role in the early detection of HD. Neurometrics is a method of statistical analysis of standardised, quantitative electrophysiological features relative to a body of normative data, developed as an aid to the differential diagnosis of a variety of subtle brain dysfunctions (25). It has been widely reported in the scientific literature (26,27) and is commercially available as the Cadwell Spectrum 32 and appropriate software.

b. THE TASMANIAN STUDY

In 1988 a long-term study into early detection of HD, using physical examination, neuropsychological testing and Neurometric 'Quantitative' (Q)EEG examination, was initiated. The plan was to compare baseline data with follow-up data, with a view to discovering early features of HD. Subjects will be reexamined at 2 yearly intervals. It is anticipated that the study will clarify the progression of neuropsychological impairment and provide details of corroborative, objective evidence of specific abnormalities of brain electrical activity. To increase yield, the subjects are at 50% risk and are between about 30 and 50 years of age. The diagnosis of HD is made only when both psychological/cognitive and physical abnormalities are present.

The psychological test battery, devised by Mr Stephen Lockwood of the University of Tasmania, after discussion with Prof Nelson Butters of the University of California (San Diego) includes, WAIS, Wechler Memory Scale, Butters Delayed Recognition Span Test, FAS letter fluency test, Category fluency test, Wisconsin Card Sorting Test and Rey Figure Test.

c. PROGRESS

To the present time, 25 examinations have been conducted. In one case neurophychological testing could not be conducted. Three individuals were found to have moderately advanced HD and two had early, but unequivocal, disease. Nineteen cases showed no abnormality or were of uncertain disease status. One individual who was in this latter category represented eighteen months later and reexamination revealed progression into the unequivocal disease category.

It is not possible to report the findings of this study as the completion of the collection of data is some years away. However, the case mentioned above is unique in the world literature and is worthy of consideration.

d. NEUROMETRICS IN EARLY HD: A CASE STUDY

(The complete results of the Neurometric analyses and comparisons of this case are detailed in Appendix II. Only highlights are presented in this chapter.)

A 26 year old, unmarried mother of three, at 50% risk for HD, presented requesting assessment of her disease status. Her mother had died of HD at 32 years of age and her brother was 28 years of age and severely affected. She wanted to determine her current disease status so that she could plan the next phase of her life (she was considering tertiary education). She complained of, among other things, lack of confidence, forgetfulness and clumsiness.

Medical examination and Neurometric QEEG was conducted. Neuropsychological testing could not be arranged. At interview the patient was composed and co-operative and, in spite of the presenting complaints, no cognitive deficit could be demonstrated using the Mini-Mental State (28). Nor was any abnormality detected on careful physical (including neurological) examination.

On Neurometric QEEG the monopolar Z score measures showed significantly reduced alpha absolute power and relative power in all regions, a significantly reduced beta absolute power in the parietal and

occipital regions, and significantly increased delta relative power in the parietal and occipital regions. See Table 1. The bipolar Z score measures showed significantly reduced alpha relative power in the central, temporal and parietal regions and increased delta relative power in the same regions. See Table 2. This is consistent with observations using traditional EEG (16-18).

It was also noted that the overall scores in the central bipolar regions (C3-CZ, C4-CZ) and the temporal bipolar regions (T3-T5, T4-T6) are significantly elevated. See Table 2.

Version 4.0 of the Psychiatric Discriminants package stated that the 'patient's Discriminants Scores lie outside the normal limits expected for and individual of this age'.

The patient returned 15 months later requesting reexamination. She complained of worsening memory and clumsiness. She said that she now disliked going to the city because she got lost and that she spilt things and bumped into people as she passed.

At interview she was still composed and co-operative, but there was moderate impairment on the 'Mini-Mental State'. Physical

Name: GK F
Age: 27.4 yrs

Analyzed: 05/30/90 Recorded: 12/02/88
Epochs: 38

Monopolar Z Score Measures

		Fp1	Fp2	F7	F8	F3	F4	C3	C4	Fpz	Fz	Cz
Absolute Power	Δ	-0.47	-0.52	-0.51	0.03	0.58	0.16	1.05	0.26	-0.42	0.42	0.6
	θ	-0.36	-0.48	-0.29	-0.35	-0.45	-0.62	-0.74	-1.14	-0.29	-0.80	-1.3
	α	-2.16	-2.15	-1.93	-1.80	-2.42	-2.32	-2.73	-2.89	-2.13	-2.55	-3.0
	β	-1.11	-0.61	-0.36	-0.39	-1.31	-0.93	-1.56	-1.14	-1.12	-1.36	-1.8
Relative Power	Δ	0.91	0.82	0.79	1.31	2.16	1.80	2.82	2.35	0.94	2.23	2.7
	θ	1.71	1.38	1.48	1.04	0.93	0.92	0.44	0.51	1.73	0.63	0.1
	α	-2.12	-2.14	-2.14	-2.27	-2.84	-2.62	-3.43	-3.32	-2.13	-2.90	-3.3
	β	0.11	0.78	0.80	0.57	-0.67	0.00	-1.03	0.16	0.06	-0.51	-0.9

Power Asymmetry	Δ	0.11	-0.65	1.05	2.25
	θ	0.40	0.14	0.52	1.44
	α	-0.02	-0.10	-0.53	0.74
	β	-1.53	0.06	-1.22	-1.41
Coherence	Δ	0.18	-0.06	0.85	1.02
	θ	0.67	0.53	0.02	0.37
	α	-1.09	-0.40	-1.33	-0.46
	β	-0.32	0.16	-0.58	0.25

		T3	T4	T5	T6	P3	P4	O1	O2	Pz	Oz
Absolute Power	Δ	-0.18	-0.06	-0.60	-1.62	0.32	-0.06	-0.62	-1.14	0.39	-1.02
	θ	-0.80	-0.51	-1.77	-2.06	-1.10	-1.29	-1.83	-2.02	-1.09	-1.91
	α	-2.15	-1.25	-3.12	-2.89	-3.00	-3.02	-2.92	-3.16	-2.98	-3.15
	β	-0.41	0.43	-2.80	-2.14	-2.27	-2.11	-2.87	-2.79	-2.10	-2.84
Relative Power	Δ	1.48	0.66	2.93	1.95	2.89	2.64	2.76	2.57	2.67	2.61
	θ	0.60	-0.12	1.07	1.30	0.98	1.19	1.21	1.36	1.01	1.41
	α	-2.43	-1.69	-3.10	-2.42	-3.41	-3.28	-2.86	-2.84	-3.38	-2.95
	β	0.73	1.51	-0.74	0.76	-0.91	-0.40	-0.47	0.11	-0.80	-0.10

Power Asymmetry	Δ	-0.03	1.65	1.05	1.82
	θ	0.07	0.68	0.43	1.19
	α	-0.32	-0.27	0.11	0.73
	β	-0.73	-1.04	-0.45	-0.07
Coherence	Δ	-2.75	-0.00	1.38	1.29
	θ	-3.55	-0.15	1.07	1.09
	α	-0.59	-2.12	-0.64	-0.18
	β	-0.55	-3.12	0.80	0.55

Table 1

Name: GK F
Age: 27.4 yrs

Analyzed: 05/30/90 Recorded: 12/02/88
Epochs: 38

Bipolar Z Score Measures

		Central		Temporal		Parietal Occipital		Frontal Temporal	
		C3/Cz	C4/Cz	T3/T5	T4/T6	P3/O1	P4/O2	F7/T3	F8/
Total Absolute Power		**-2.11**	-1.47	-1.62	-0.86	-1.76	-1.78	-1.31	-0.
Total Power Asymmetry		-1.61		-1.58		-0.06		-1.11	
Relative Power	Δ	1.72	**2.83**	1.48	**2.07**	**2.35**	**2.48**	0.26	-0.
	θ	1.05	0.21	0.86	0.55	1.68	1.63	0.55	0.
	α	**-2.25**	**-2.95**	**-2.60**	**-2.94**	**-2.66**	**-2.65**	-1.82	-1.
	β	-0.11	-0.26	1.54	1.61	0.11	0.19	1.34	**1.**
	Δ+θ	1.88	**2.44**	1.39	1.71	**2.69**	**2.71**	0.34	-0.
	Combination	1.06	**2.41**	1.37	**2.12**	1.42	1.44	0.78	1.
Power Asymmetry	Δ	**-2.73**		**-2.15**		-0.08		-0.43	
	θ	-0.58		-1.21		-0.05		-0.56	
	α	0.03		-0.92		-0.12		-0.69	
	β	-1.24		-1.38		-0.15		-1.07	
	Combination	1.42		0.80		-4.22		-0.75	
Coherence	Δ	-0.18		0.07		1.22		-0.22	
	θ	-1.88		-0.36		0.47		0.17	
	α	-0.55		-1.81		-0.67		-1.20	
	β	0.60		**-3.62**		-1.12		**-2.03**	
	Combination	0.96		1.92		0.38		1.00	
Overall		**-2.39**		**-2.77**		0.74		0.52	

Table 2

Name: GK2 F
Age: 28.9 yrs

Analyzed: 06/12/90
Epochs: 38

Recorded: 05/23/90
Site Id: RHH

Monopolar Z Score Measures

		Fp1	Fp2	F7	F8	F3	F4	C3	C4	Fpz	Fz	Cz
Absolute Power	Δ	0.69	0.54	-1.95	0.93	-2.39	1.73	-2.63	1.93	0.91	-2.05	-1.98
	θ	-0.41	-0.45	0.29	-0.41	-0.19	-0.52	-0.01	-0.48	-0.37	-0.65	-0.73
	α	-2.63	-2.43	-2.18	-2.14	-2.64	-2.63	-2.72	-2.77	-2.40	-2.78	-2.91
	β	-2.30	-1.04	-0.61	-1.16	-1.78	-1.54	-2.09	-1.84	-1.70	-2.14	-2.31
Relative Power	Δ	2.43	2.01	2.91	2.58	3.60	3.27	3.74	3.50	2.47	3.68	3.61
	θ	0.60	0.52	0.22	0.40	-0.22	-0.10	-0.01	-0.01	0.27	-0.52	-0.35
	α	-3.13	-2.88	-3.51	-2.99	-3.83	-3.58	-4.20	-3.95	-3.01	-3.85	-3.92
	β	-1.87	-0.38	-1.04	-0.87	-2.29	-1.62	-2.79	-1.91	-1.43	-2.50	-2.65

Power Asymmetry	Δ	0.63	1.26	1.49	1.84
	θ	0.12	1.02	0.97	1.57
	α	-1.29	0.15	-0.05	0.28
	β	-3.82	0.89	-0.77	-0.97
Coherence	Δ	2.16	1.62	2.49	2.97
	θ	1.63	1.16	1.09	1.94
	α	-1.12	-1.04	-1.23	0.56
	β	-1.20	-0.68	-0.95	1.40

		T3	T4	T5	T6	P3	P4	O1	O2	Pz	Oz
Absolute Power	Δ	-2.37	0.69	1.68	1.25	1.86	1.85	-2.40	1.75	1.47	1.33
	θ	0.33	-0.28	-0.48	-0.60	-0.39	-0.50	-0.28	-0.17	-0.99	-0.60
	α	-1.98	-1.24	-2.48	-2.13	-2.64	-2.57	-2.42	-2.46	-2.65	-2.35
	β	-0.97	-0.16	-2.07	-1.16	-2.28	-1.93	-2.02	-1.67	-2.04	-1.62
Relative Power	Δ	-3.54	1.88	3.66	3.08	3.54	3.44	3.75	3.36	3.23	3.13
	θ	0.33	0.02	0.62	0.67	0.54	0.54	0.56	1.01	0.31	0.88
	α	-3.40	-1.87	-3.51	-2.97	-3.69	-3.60	-3.71	-3.56	-3.37	-3.15
	β	-1.44	0.16	-1.63	-0.30	-2.05	-1.58	-1.64	-0.92	-1.42	-0.51

Power Asymmetry	Δ	0.59	0.39	0.16	1.31
	θ	0.52	0.23	0.27	-0.26
	α	-0.18	-0.65	-0.21	0.02
	β	-0.50	-1.63	-1.01	-0.86
Coherence	Δ	1.75	2.09	2.65	1.25
	θ	1.62	2.11	2.32	2.30
	α	-1.58	0.93	1.09	1.83
	β	-1.12	1.77	2.11	3.36

Table 3

Name: GK2 F
Age: 28.9 yrs

Analyzed: 06/12/90
Epochs: 38

Recorded: 05/23/90
Site Id: RHH

Bipolar Z Score Measures

		Central		Temporal		Parietal Occipital		Frontal Temporal	
		C3/Cz	C4/Cz	T3/T5	T4/T6	P3/O1	P4/O2	F7/T3	F8/T
Total Absolute Power		**-2.36**	-1.82	-1.76	-1.29	-1.37	**-2.50**	-1.38	-1.2
Total Power Asymmetry		-1.33		-0.90		**2.78**		-0.22	
Relative Power	Δ	**2.51**	**3.63**	1.82	**2.42**	**4.05**	**2.45**	0.22	0.1
	θ	0.96	0.23	0.94	0.51	0.50	1.52	0.62	0.3
	α	**-2.73**	**-3.25**	**-2.06**	**-2.38**	**-3.94**	**-2.75**	-1.89	-1.5
	β	-1.11	-1.79	0.87	0.98	-1.47	0.49	1.39	1.5
	Δ+θ	**2.72**	**3.50**	1.73	**2.01**	**4.24**	**2.58**	0.33	0.1
	Combination	1.72	**2.80**	0.94	**2.00**	**3.20**	1.54	0.89	0.7
Power Asymmetry	Δ	**-2.26**		-1.45		**3.71**		0.03	
	θ	-0.43		-0.38		1.38		0.15	
	α	-0.04		-0.39		0.11		-0.63	
	β	-0.15		-0.86		-0.53		-0.26	
	Combination	0.95		0.45		**2.12**		-1.39	
Coherence	Δ	0.19		0.13		**-2.36**		-0.59	
	θ	**-3.51**		-0.42		**-2.17**		0.44	
	α	-0.62		-1.25		**-2.42**		-1.73	
	β	0.74		-1.52		-1.20		**-2.58**	
	Combination	**2.49**		0.09		1.55		1.83	
Overall		**4.10**		0.91		**3.53**		1.38	

Table 4

Name 1: GK1 F
ta is GK1 compared to GK2 Name 2: K Gina
nopolar Independent Tscore/Diff. Zscore Measures Analyzed: 06/12/90
grees of freedom: 74

		Fp1	Fp2	F7	F8	F3	F4	C3	C4	Fpz	Fz	Cz
bsolute	Δ	-3.6	-2.9	-5.9	-2.4	-4.1	-3.3	-3.9	-3.9	-4.2	-3.4	-3.7
Power	θ	0.2	-0.0	-2.2	0.3	-0.9	-0.3	-2.9	-2.5	0.4	-2.3	-0.4
Tscore)	α	3.7	2.2	1.8	2.2	1.7	2.5	-0.2	-1.0	2.1	-0.4	1.9
	ß	5.7	1.9	1.1	5.0	2.2	3.5	2.8	3.6	2.6	3.1	3.7
elative	Δ	-1.5	-1.2	-2.1	-1.3	-1.4	-1.5	-0.9	-1.2	-1.5	-1.4	-0.8
Power	θ	1.1	0.9	1.3	0.6	1.1	1.0	0.5	0.5	1.5	1.1	0.5
(Δ Z)	α	1.0	0.7	1.4	0.7	1.0	1.0	0.8	0.6	0.9	0.9	0.6
	ß	2.0	1.2	1.8	1.4	1.6	1.6	1.8	2.1	1.5	2.0	1.7

Power	Δ	-0.5	-1.9	-0.4	0.4
ymmetry	θ	0.3	-0.9	-0.5	-0.1
(Δ Z)	α	1.3	-0.2	-0.5	0.5
	ß	2.3	-0.8	-0.4	-0.4
herence	Δ	-2.0	-1.7	-1.6	-1.9
(Δ Z)	θ	-1.0	-0.6	-1.1	-1.6
	α	0.0	0.6	-0.1	-1.0
	ß	0.9	0.8	0.4	-1.1

		T3	T4	T5	T6	P3	P4	O1	O2	Pz	Oz
bsolute	Δ	-6.2	-4.0	-6.4	-8.1	-4.4	-5.0	-8.0	-7.5	-2.9	-6.7
Power	θ	-5.0	-1.7	-6.2	-6.3	-3.1	-3.2	-6.6	-7.8	-0.3	-6.5
Tscore)	α	-1.2	-0.1	-4.3	-4.9	-2.6	-3.0	-2.2	-3.3	-2.2	-3.4
	ß	2.9	3.9	-6.9	-8.0	-0.2	-1.6	-7.0	-7.7	-0.6	-8.8
elative	Δ	-2.1	-1.2	-0.7	-1.1	-0.6	-0.8	-1.0	-0.8	-0.6	-0.5
Power	θ	0.3	-0.1	0.5	0.6	0.4	0.6	0.7	0.3	0.7	0.5
(Δ Z)	α	1.0	0.2	0.4	0.6	0.3	0.3	0.9	0.7	-0.0	0.2
	ß	2.2	1.4	0.9	1.1	1.1	1.2	1.2	1.0	0.6	0.4

Power	Δ	-0.6	1.3	0.9	0.5
ymmetry	θ	-0.5	0.5	0.2	1.5
(Δ Z)	α	-0.1	0.4	0.3	0.7
	ß	-0.2	0.6	0.6	0.8
herence	Δ	-4.5	-2.1	-1.3	0.0
(Δ Z)	θ	-5.2	-2.3	-1.2	-1.2
	α	1.0	-3.1	-1.7	-2.0
	ß	0.6	-4.9	-1.3	-2.8

Table 5

Name 1: GK1 F
Name 2: K

Data is GK1 compared to GK2
Bipolar Independent Tscore/Diff. Zscore Measures Analyzed: 06/12/90
Degrees of freedom: 74

		Central		Temporal		Parietal Occipital		Frontal Temporal	
		C3/Cz	C4/Cz	T3/T5	T4/T6	P3/O1	P4/O2	F7/T3	F8/T4
Absolute Power (Tscore)	Δ	-0.7	0.2	-0.2	1.5	-6.1	4.0	0.6	1.3
	θ	1.7	1.8	0.9	3.3	1.8	5.3	0.4	2.0
	α	3.0	2.6	-2.4	-0.3	1.9	2.5	0.7	0.4
	β	6.3	6.5	3.5	4.2	6.4	5.3	0.1	3.3
Relative Power (Δ Z)	Δ	-0.8	-0.8	-0.3	-0.3	-1.7	0.0	0.0	-0.2
	θ	0.1	-0.0	-0.1	0.0	1.2	0.1	-0.1	-0.3
	α	0.5	0.3	-0.5	-0.6	1.3	0.1	0.1	-0.3
	β	1.0	1.5	0.7	0.6	1.6	-0.3	-0.0	0.4
Power Asymmetry (Δ Z)	Δ	-0.5		-0.7		-3.8		-0.5	
	θ	-0.1		-0.8		-1.4		-0.7	
	α	0.1		-0.5		-0.2		-0.1	
	β	-1.1		-0.5		0.4		-0.8	
Coherence (Δ Z)	Δ	-0.4		-0.1		3.6		0.4	
	θ	1.6		0.1		2.6		-0.3	
	α	0.1		-0.6		1.8		0.5	
	β	-0.1		-2.1		0.1		0.5	

Table 6

Name 1: GK1 F
ata is GK1 compared to GK2 Name 2:
onopolar Correlated Tscore/Diff. Zscore Measures Analyzed: 06/12/90
egrees of freedom: 37

		Fp1	Fp2	F7	F8	F3	F4	C3	C4	Fpz	Fz	Cz
bsolute	Δ	-3.7	-2.9	-6.2	-2.5	-4.3	-3.4	-4.0	-4.0	-4.3	-3.4	-3.8
Power	θ	0.3	-0.0	-2.4	0.3	-0.9	-0.3	-3.1	-2.9	0.4	-2.5	-0.5
Tscore)	α	3.5	2.1	1.7	2.1	1.5	2.2	-0.2	-0.9	1.9	-0.4	1.6
	β	5.7	1.9	1.1	4.8	2.5	3.6	2.8	3.3	3.1	3.2	3.9
elative	Δ	-1.5	-1.2	-2.1	-1.3	-1.4	-1.5	-0.9	-1.2	-1.5	-1.4	-0.8
Power	θ	1.1	0.9	1.3	0.6	1.1	1.0	0.5	0.5	1.5	1.1	0.5
(Δ Z)	α	1.0	0.7	1.4	0.7	1.0	1.0	0.8	0.6	0.9	0.9	0.6
	β	2.0	1.2	1.8	1.4	1.6	1.6	1.8	2.1	1.5	2.0	1.7

Power	Δ	-0.5	-1.9	-0.4	0.4
ymmetry	θ	0.3	-0.9	-0.5	-0.1
(Δ Z)	α	1.3	-0.2	-0.5	0.5
	β	2.3	-0.8	-0.4	-0.4
herence	Δ	-2.0	-1.7	-1.6	-1.9
(Δ Z)	θ	-1.0	-0.6	-1.1	-1.6
	α	0.0	0.6	-0.1	-1.0
	β	0.9	0.8	0.4	-1.1

		T3	T4	T5	T6	P3	P4	O1	O2	Pz	Oz
bsolute	Δ	-6.2	-4.0	-6.4	-8.1	-4.5	-5.0	-8.3	-7.6	-3.0	-7.0
Power	θ	-5.3	-1.8	-5.9	-7.6	-3.3	-4.0	-6.5	-8.3	-0.3	-6.4
Tscore)	α	-1.1	-0.1	-4.3	-5.3	-2.6	-3.1	-2.3	-3.5	-2.4	-3.5
	β	3.3	5.3	-6.6	-8.7	-0.2	-1.4	-6.4	-7.6	-0.6	-8.1
elative	Δ	-2.1	-1.2	-0.7	-1.1	-0.6	-0.8	-1.0	-0.8	-0.6	-0.5
Power	θ	0.3	-0.1	0.5	0.6	0.4	0.6	0.7	0.3	0.7	0.5
(Δ Z)	α	1.0	0.2	0.4	0.6	0.3	0.3	0.9	0.7	-0.0	0.2
	β	2.2	1.4	0.9	1.1	1.1	1.2	1.2	1.0	0.6	0.4

Power	Δ	-0.6	1.3	0.9	0.5
ymmetry	θ	-0.5	0.5	0.2	1.5
(Δ Z)	α	-0.1	0.4	0.3	0.7
	β	-0.2	0.6	0.6	0.8
herence	Δ	-4.5	-2.1	-1.3	0.0
(Δ Z)	θ	-5.2	-2.3	-1.2	-1.2
	α	1.0	-3.1	-1.7	-2.0
	β	0.6	-4.9	-1.3	-2.8

Table 7

Name 1: GK1 F
ata is GK1 compared to GK2 Name 2: K
ipolar Correlated Tscore/Diff. Zscore Measures Analyzed: 06/12/90
egrees of freedom: 37

		Central		Temporal		Parietal Occipital		Frontal Temporal	
		C3/Cz	C4/Cz	T3/T5	T4/T6	P3/O1	P4/O2	F7/T3	F8/T4
Absolute Power (Tscore)	Δ	-0.7	0.2	-0.2	1.6	-6.3	3.7	0.6	1.3
	θ	1.6	2.1	0.9	3.0	2.0	5.2	0.3	2.0
	α	2.7	2.4	-2.3	-0.3	1.9	2.4	0.7	0.4
	ß	6.9	6.0	3.8	5.2	6.4	5.0	0.1	4.6
Relative Power (Δ Z)	Δ	-0.8	-0.8	-0.3	-0.3	-1.7	0.0	0.0	-0.2
	θ	0.1	-0.0	-0.1	0.0	1.2	0.1	-0.1	-0.3
	α	0.5	0.3	-0.5	-0.6	1.3	0.1	0.1	-0.3
	ß	1.0	1.5	0.7	0.6	1.6	-0.3	-0.0	0.4

Power Symmetry (Δ Z)	Δ	-0.5	-0.7	-3.8	-0.5
	θ	-0.1	-0.8	-1.4	-0.7
	α	0.1	-0.5	-0.2	-0.1
	ß	-1.1	-0.5	0.4	-0.8
Coherence (Δ Z)	Δ	-0.4	-0.1	3.6	0.4
	θ	1.6	0.1	2.6	-0.3
	α	0.1	-0.6	1.8	0.5
	ß	-0.1	-2.1	0.1	0.5

Table 8

examination revealed involuntary movements of the fingers, difficulty with rapidly alternating movements, stiff gait, difficulty with standing with eyes closed, and increased reflexes in the left arm and both legs, with Babinski's upgoing on both sides. Neuropsychological testing revealed gross impairment.

On repeat Neurometric analysis the results were similar to those of the first examination but the deviation from normative values was greater. In the monopolar Z score measures there was reduced absolute and relative alpha power in every region, increased delta relative power in every region and increased coherence in various regions. See Table 3. In the bipolar Z score measures there was reduced alpha relative power in the central, temporal and parietal regions, and increased delta relative power in the same regions. There was significant coherence in various regions. See Table 4.

The overall scores in the central bipolar region (C3-CZ, C4-CZ) were very significantly elevated. See Table 4.

Version 4.0 of the Psychiatric Discriminants package made the same comment as on the previous analysis.

The two sets of results were compared (monopolar and bipolar, independent and correlated) using T scores of absolute power and Z scores of relative power, power assymetry and coherence. See Tables 5 to 8. Analysis using the monopolar correlated T scores reveals a significant increase in delta absolute power over the entire cortex and a significant increase in theta absolute power in the temporal, parietal and occipital regions. There is a decrease in beta activity in all regions except in the parietal and occipital regions. There is also a tendency toward increased temporal-parietal-occipital coherence (with many values being statistically significant) indicating an increase in the synchronicity of the EEG in these areas in the second assessment that was not present in the first. See Table 7. The bipolar correlated data shows a significantly decreased beta absolute power across the entire cortex. See Table 8.

Summarising the Neurometric results, there was an initial decline in alpha absolute and relative power which was fairly generalised across the cortex (observed in the first assessment), which was followed by a continuing decrease in alpha and beta activity with a concomitant increase in delta and theta activity (observed in the second assessment). These changes were present in both monopolar and bipolar studies.

This pattern is consistent with current knowledge of

dementing disorders. It is an orderly progression of observations. Accordingly, it may be presumed that the changes observed at the initial Neurometric examination of this case were the earliest obtainable neurophysiological evidence of the disorder. Thus, Neurometric QEEG may have a place in the early detection of HD.

The overall scores in the central bipolar regions (C3-CZ, C4-Cz) provide a multivariate indication of abnormality and may become the most useful single neurphysiological observation in predicting a decline in function (clinical diagnosis).

A comprehensive study of the the value of Neurometrics in the early detection of HD is in progress.

e. REFERENCES

1. Turner D, Haan E, Jacka E, Kalucy R, Burns R, Willoughby J, Crabb R. Prenatal and adult presymptomatic testing for Huntington's disease. Medical Journal of Australia 1988 ; 148: 567-573.

2. Gusella J, Wexler N, Conneally P. A polymorphic DNA marker genetically linked to Huntington's disease. Nature 1983; 306: 234-238.

3. Markel D, Young A, Penney J, et al. At risk person's attitudes toward presymptomatic and prenatal testing for Huntington's disease in Michigan American Journal of Medical Genetics 1987; 26: 295-305.

4. Simpson S, Johnston A. The prevalence and patterns of care of Huntington's chorea in Grampian. British Journal of Psychiatry 1989; 155: 799-804.

5. Quaid K, Brandt J, Folstein S. The decision to be tested for Huntington's chorea. Journal of American Medical Association 1987; 257: 3362-3367.

6. Pridmore S. Age of onset in Huntiogton's disease in Tasmania. Medical Journal of Australia 1990; 153: 135-137.

7. Goodman R, Hall C, Terrango L, et al. Huntington's chorea: A multidisciplinary study of affected parents and first generation offspring. Arch Neurol 1966; 15: 345-355.

8. Josiannen R, Curry L, Mancall E. Develpement of neuropsychological deficits in Huntington's disease. Arch Neurol 1983; 40: 791-796.

9. Baro F. A neuropsychological approach to early detection of Huntington's chorea. Adv Neurol 1973: 1: 329-338.

10. Oepen G, Mohr U, Williams K, et al. Huntington's disease: Visuomotor disturbance in patients and offspring. J Neurol Neurosurg Psychiatry 1985; 48:426-433.

11. Wexler N. Perceptual-motor, cognitive, and emotional characteristics of persons at risk for Huntington's disease. Adv Neurol 1978; 23: 257-271.

12. Fido P, Cox C, Neophytides A, et al. Neuropsychological profile of Huntington's disease: Patients and those at risk. Adv Neurol 1979; 23: 239-255.

13. Jason G, Pajurkova E, Suchowersky O, et al. Presymptomatic neuropsychological impairment in Huntington's disease. Arch Neurol 1988; 45: 769-773.

14. Hayden M, Hewitt J, Stoessl A, et al. The combined use of positron emission tomography and DNA polymorphisms for preclinical detection of Huntington's disease. Neurology 1987; 37: 1441-1447.

15. Folstein S. Huntington's disease. Baltimore: The John Hopkins University Press, 1989: 145.

16. Hill D. Proc R Soc Med 1948. 41; 242-252.

17. Margerison J, Scott D. Huntington's chorea; clinical, EEG and neuropathological findings. Electroenceph clin Neurophysiol 1965; 19: 314.

18. Scott D, Heathfield K, Toone B, Margerison J. The EEG in Huntington's chorea: a clinical and neuropathological study. J Neurol Neurosurg Psychiatry 1972; 35: 97-102.

19. Hayden M. Huntington's chorea. New York: Springer-Verlag, 1981: 81.

20. Oliver J. Huntington's chorea in Northhamptonshire. Brit J Psychiat 1979; 116: 241-253.

21. Heathfield K. Huntington's chorea. Brain 1967; 90: 203-232.

22. Patterson R, Bagchi B, Test A. The prediction of Huntington's chorea. American Journal of Psychiatry1948; 104:786-797.

23. Prichep L, Gomez-Mont F, John E, Ferris S. Neurometric electroencephalographic characteristics of dementia. In: B Reisberg ed. Alzheimer's disease. The standard reference. New York: Free Press, 1983.

24. Leuchter A, Spar J, Walter D, Weiner H. Electroencephalographic spectra and coherence in the diagnosis of Alzheimer's-type and multi-infarct dementia. Arch Gen Psychiatry 1987; 44: 993-998.

25. John E, Karmel B, Corning W, Easton P, Ahn H, Harmeny T, Prichep L, Toro A, Gerson I, Bartlett F, Thatcher R, Kaye H Valdes P, Schwartze E. Neurometrics: numerical taxonomy identifies different profiles of the brain functions within groups of behaviorally similar people. Science 1977; 196: 1393-1410.

26. John E, Prichep L, Fridman J, Easton P. Neurometric topographic mapping of EEG and evoked potenial features: Application to clinical diagnosis and cognitive evaluation. In: Maurer K. ed. Topographic Brain Mapping of EEG and Evoked Potentials. Berlin: Springer-Verlag, 1989: 90-117.

27. John E, Prichep L, Fridman J, Easton P. Neurometrics: computer-assisted differential diagnosis of brain Dysfunction. Science 1988; 239:162-169.

28. Folstein M, Folstein S, McHugh P. 'Mini-Mental State': A practical method for grading the sognitive state of patients for the clinician. Journal of Psychiatric Research 1975; 2: 189-198.

APPENDIX I

THE PEDIGREE OF THE LARGE HUNTINGTON'S DISEASE FAMILY OF TASMANIA

This is an appendix to Chapter 10. It is the full pedigree of the Brothers family; it illustrates the relationships of 1568 individuals over nine generations. It occupies the next 152 pages.

The first step in the producing this pedigree was the collection of the data. This is described in Chapter 4.

The second step was to enter this data on an IBM compatible PC using a programme known as the Huntington's Chorea Clinical Register System (V2.2). This was obtained from the Institute of Medical Genetics, University of Wales College of Medicine, Cardiff.

The final step was printing, using the programme PLOT2000. This was also obtained from the Institute of Medical Genetics, University of Wales College of Medicine. This programme is accessed via the Huntington's Chorea Clinical Register System (V2.2) main menu.

In this example, each person is identified by a generation

and an individual number. Where known, the date of birth has also been added. Otherwise, conventional symbols have been employed.

APPENDIX II

NEUROMETRIC MAPS AND MEASURES: A CASE STUDY

This is an appendix to Chapter 12. It is a complete collection of neurometric maps and measures obtained from two examinations of the same patient, the first on 2/12/1988 and the second on 25/5/1990. In addition, it contains full details of a comparison of these sets of results.

The hardware used was a Cadwell Spectrum 32; the programme, was Psychiatric Discriminants, Version 4. The comparison was made using the Stored T-score data analysis option. It should be noted that the comparison of absolute power is defined in t-test units, and that of relative power, power asymmetry, and coherence are defined in Z-score units. T-test tables and Z score tables are required for evaluation.

The maps and measures are arranged in the following order:

<u>From 2/12/1988</u>

1. Neurometric Analysis Discriminants
2. Monopolar Raw Measures
3. Monopolar Raw Maps
4. Monopolar Normative Measures
5. Monopolar Normative Maps
6. Monopolar Z Score Measures

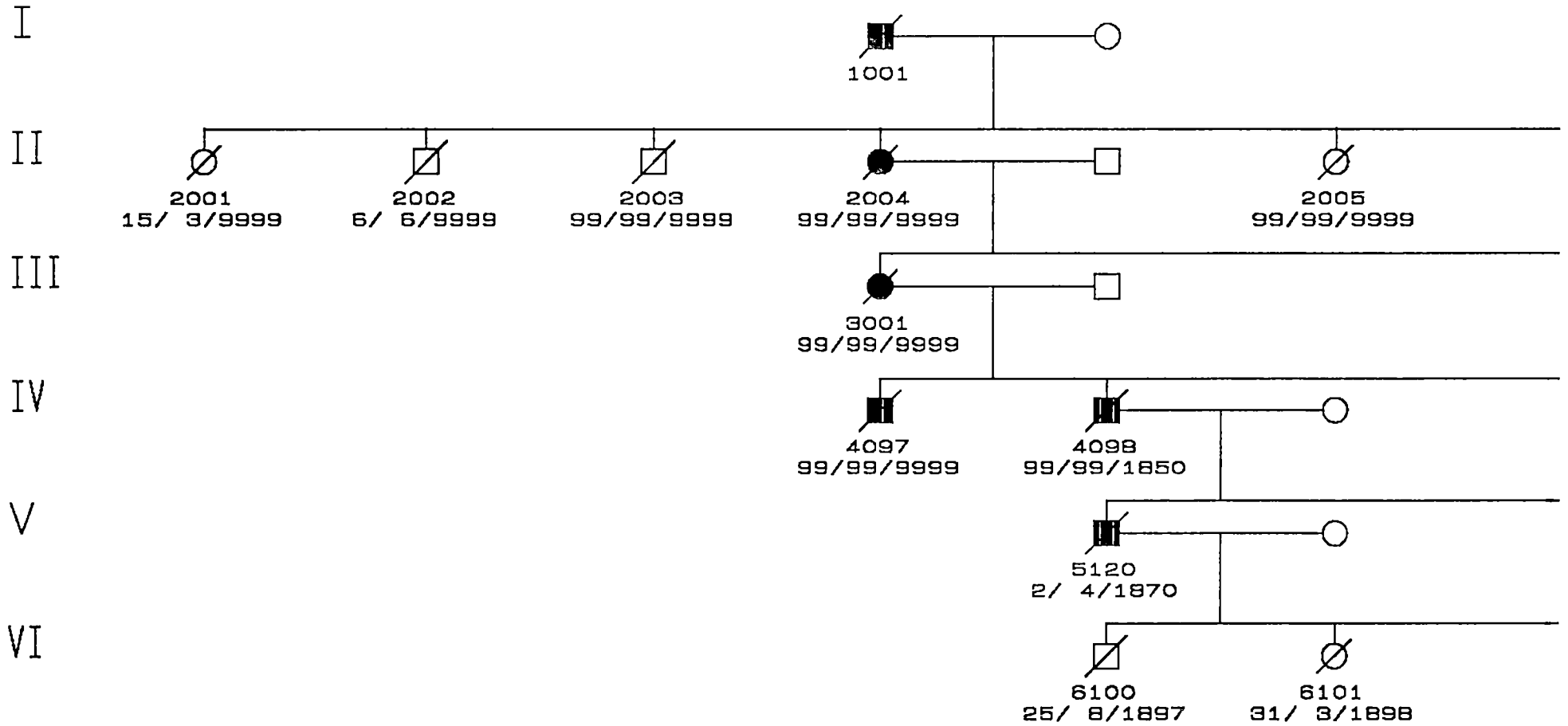
7. Monopolar Z Score Maps
8. Bipolar Raw Measures
9. Bipolar Raw Maps
10. Bipolar Normative Measures
11. Bipolar Normative Maps
12. Bipolar Z Score Measures
13. Bipolar Z Score Maps
14. Bipolar Multivariate Z Score Measures

From 25/5/1990

15. Neurometric Analysis Discriminants
16. Monopolar Raw Measures
17. Monopolar Raw Maps
18. Monopolar Raw Measures
19. Monopolar Normative Maps
20. Monopolar Z Score Measures
21. Monopolar Z Score Maps
22. Bipolar Raw Measures
23. Bipolar Raw Maps
24. Bipolar Normative Measures
25. Bipolar Normative Maps
26. Bipolar Z Score Measures
27. Bipolar Z Score Maps
28. Bipolar Multivariate Z Score Measures

Comparison of the results of 2/12/1990 and 25/5/1990

29. Monopolar Independent Tscore/Diff. Zscore Measures
30. Monopolar Independent Tscore/Diff. Zscore Maps
31. Bipolar Independent Tscore/Diff. Zscore Measures
32. Bipolar Independent Tscore/Diff. Zscore Maps
33. Monopolar Correlated Tscore/Diff. Zscore Measures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IV
V
VI
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6/ 6/9999
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2005
99/99/9999
3001
99/99/9999
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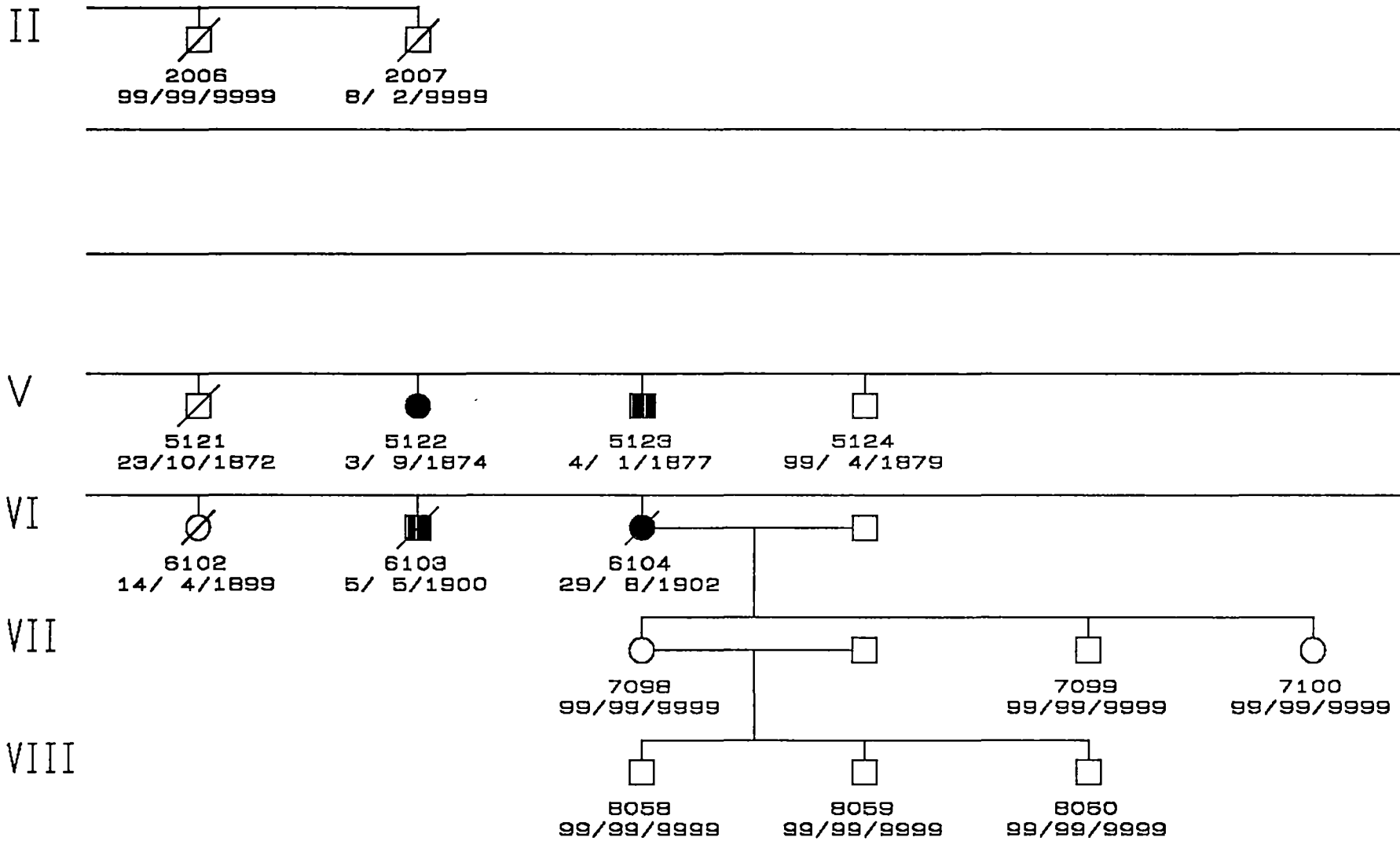
2/ 4/1870
6100
25/ 8/1897
6101
31/ 3/1898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8/ 2/9999
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4/ 1/1877
5124
99/ 4/1879
VI
6102
14/ 4/1899
6103
5/ 5/1900
6104
29/ 8/1902
VII
7098
99/99/9999
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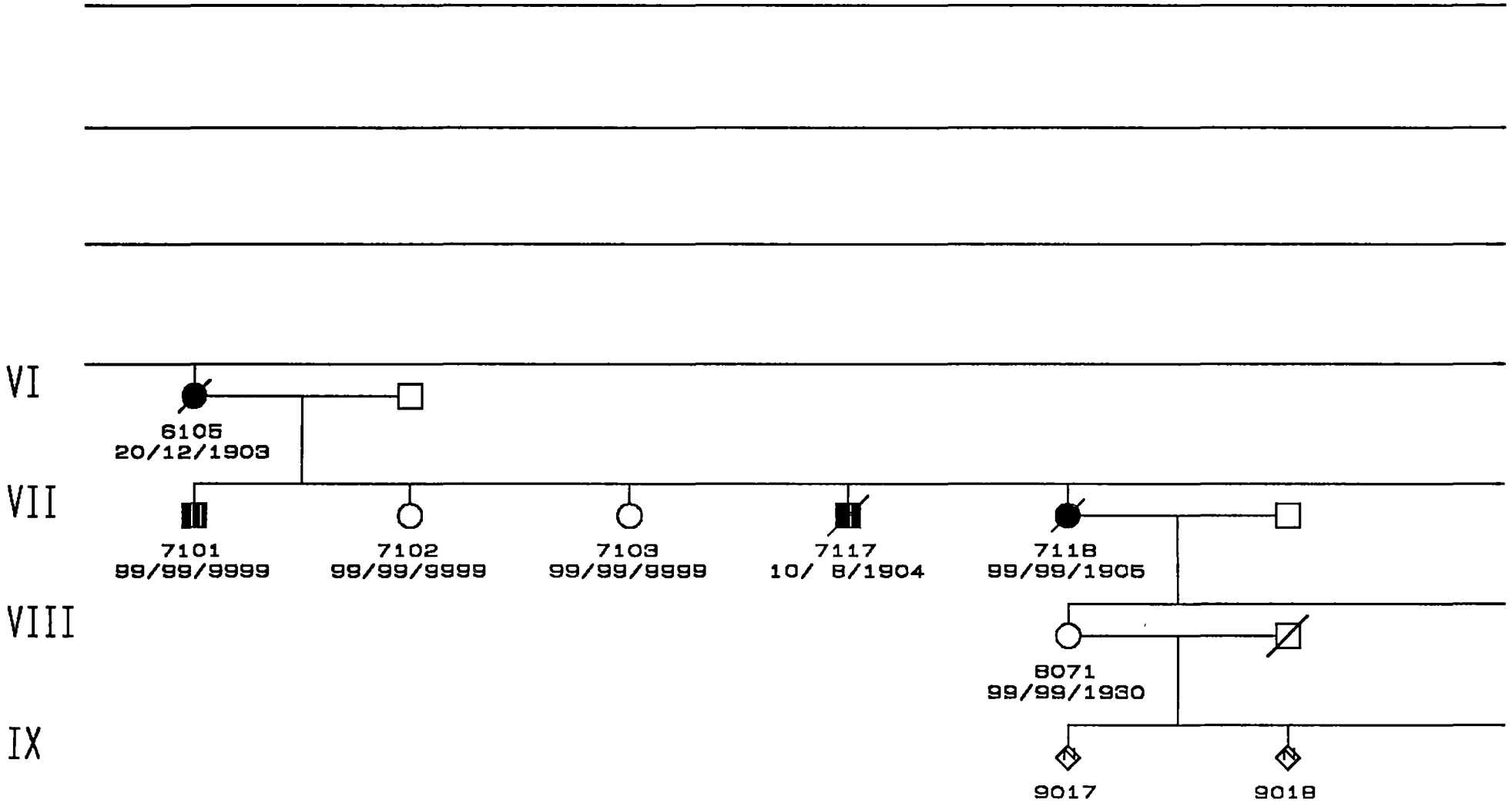
99/99/9999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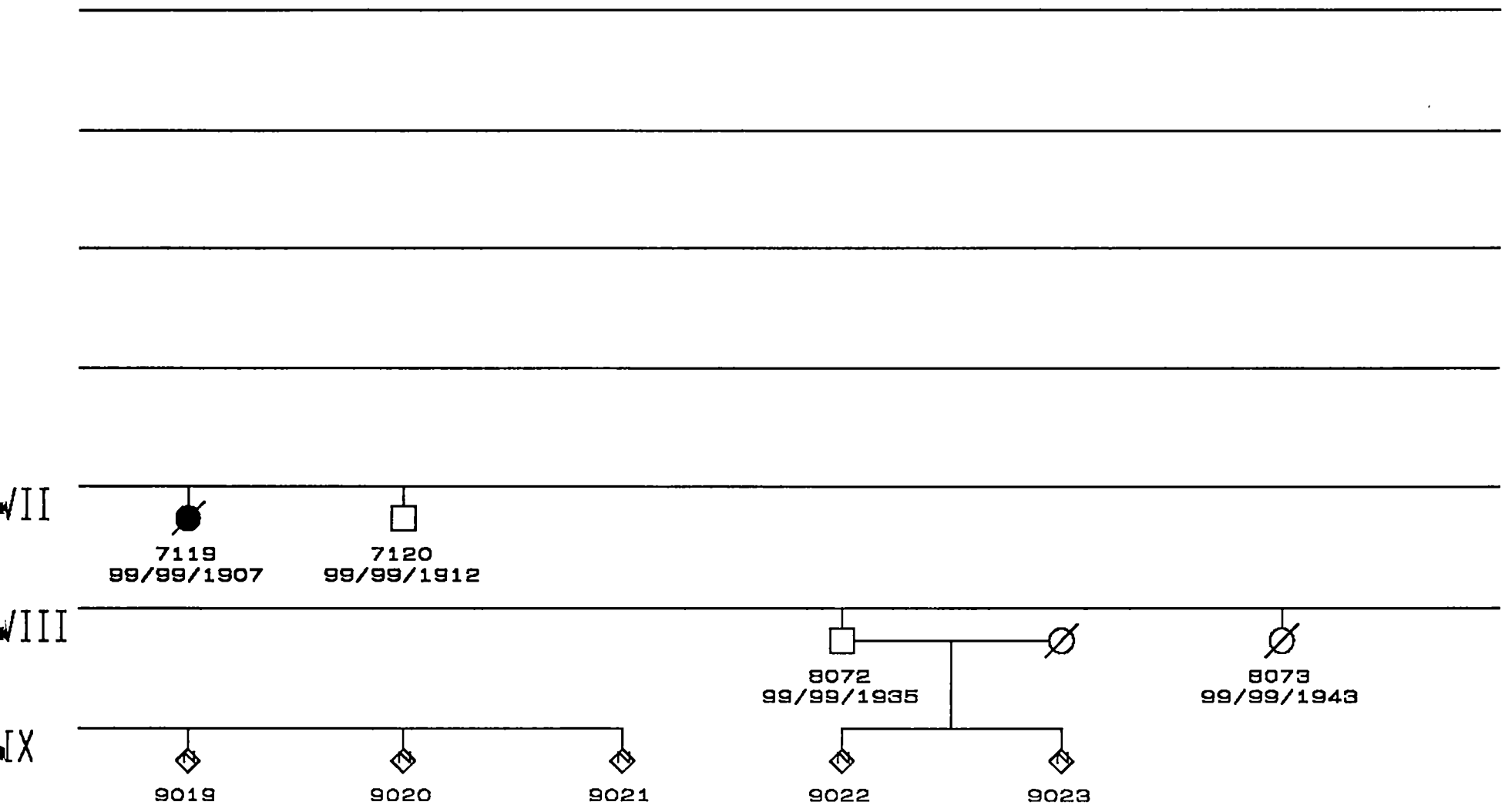
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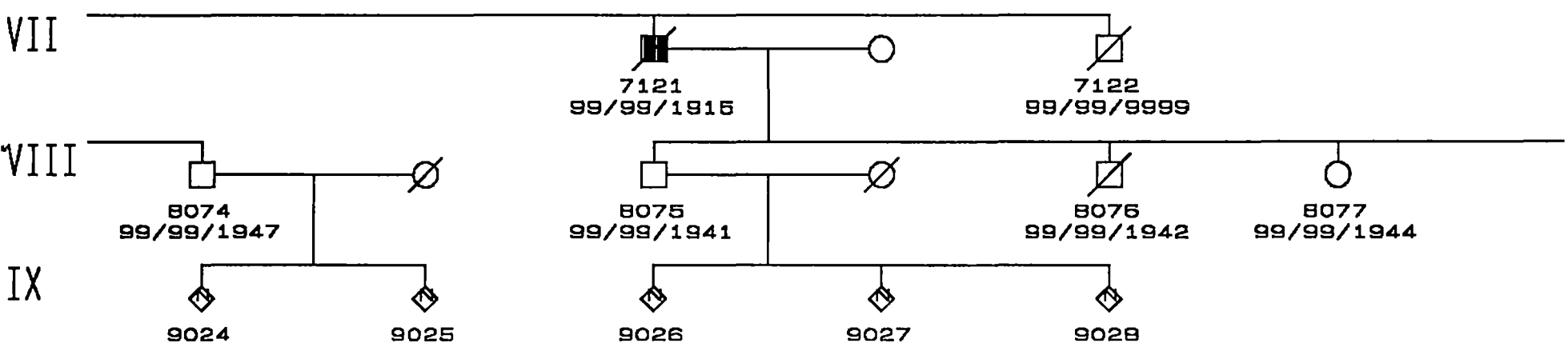
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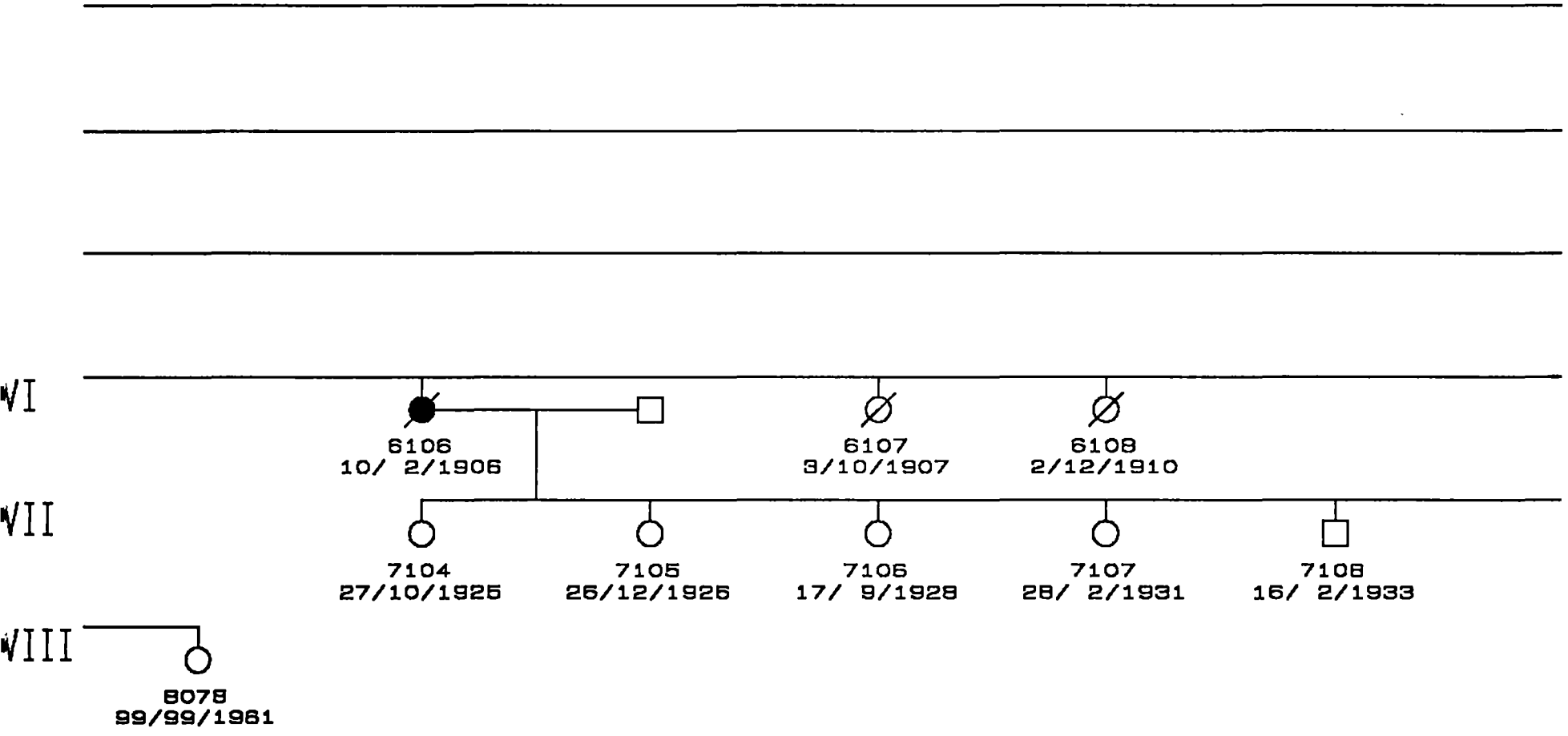
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VIII
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3/10/1907
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27/10/1925
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28/ 2/1931
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16/ 2/1933
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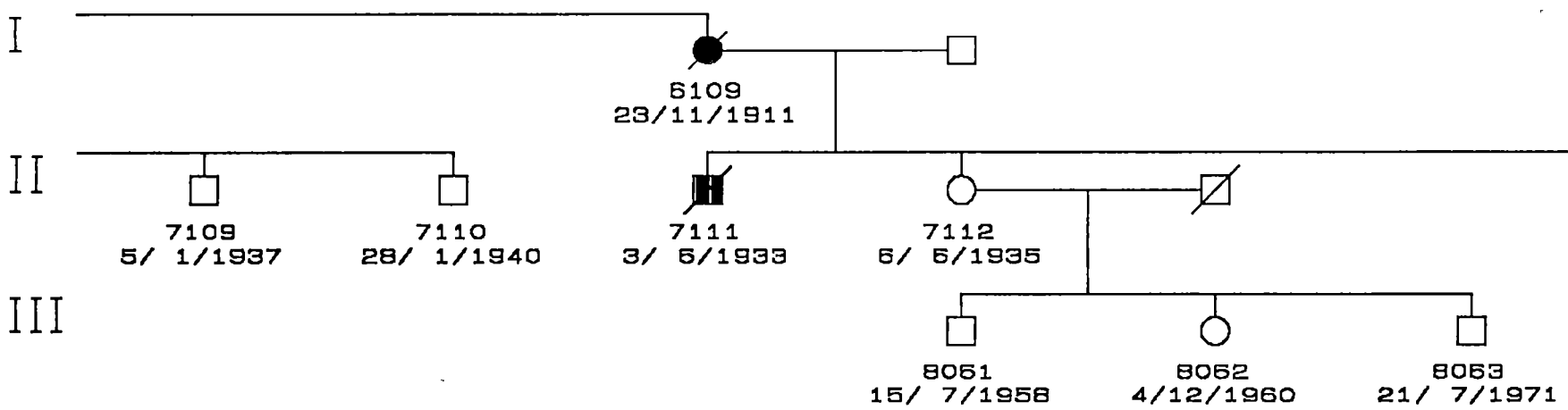
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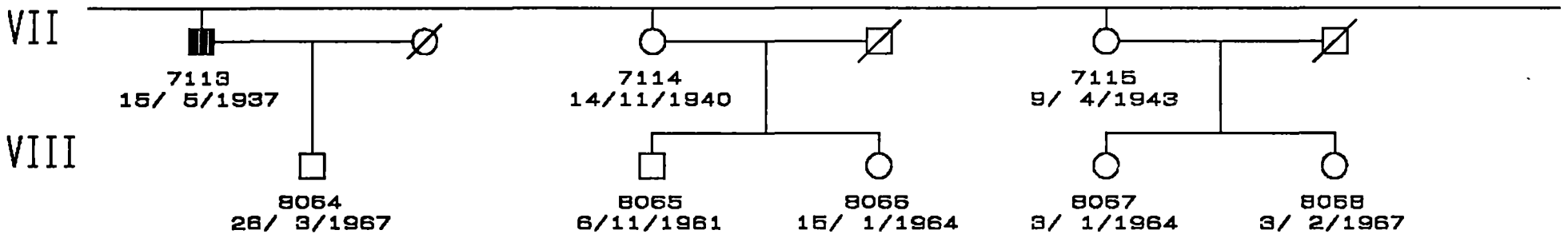
21/ 7/1971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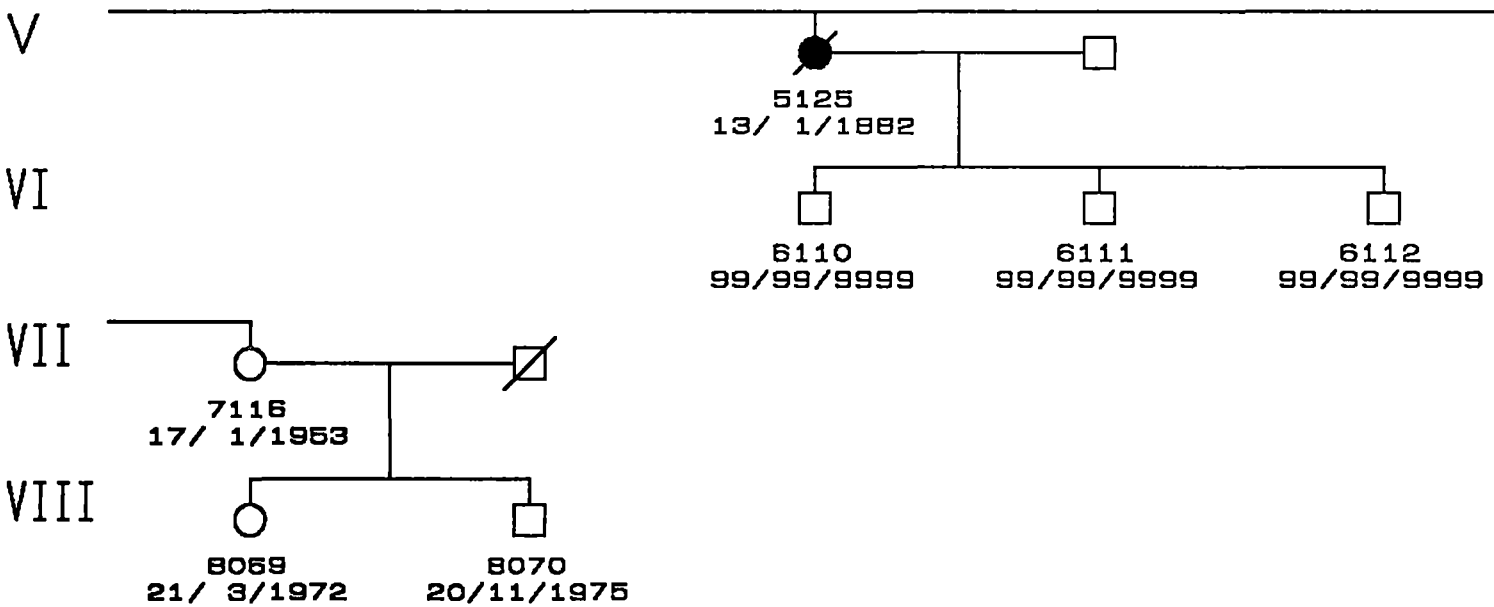
3/ 2/1967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21/ 3/1972
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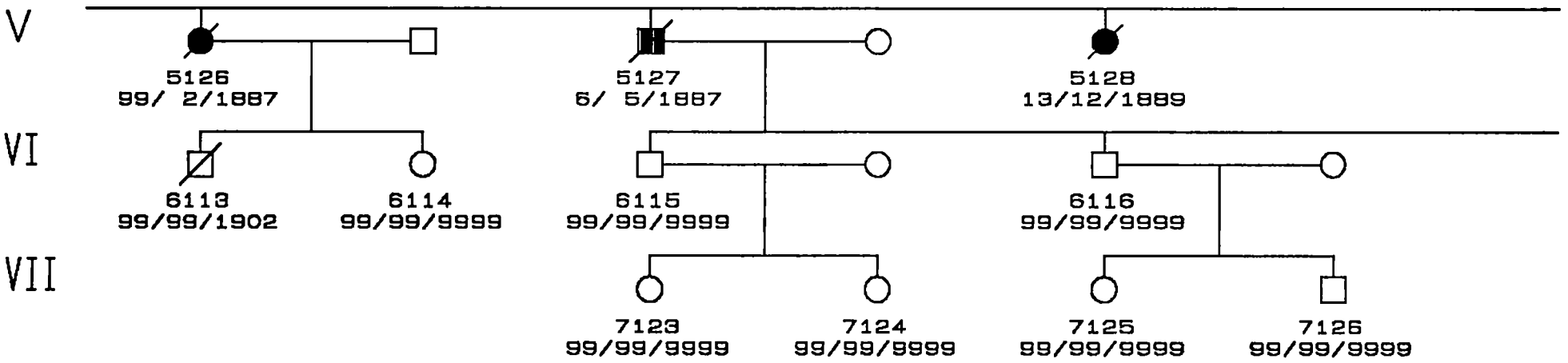
20/11/1975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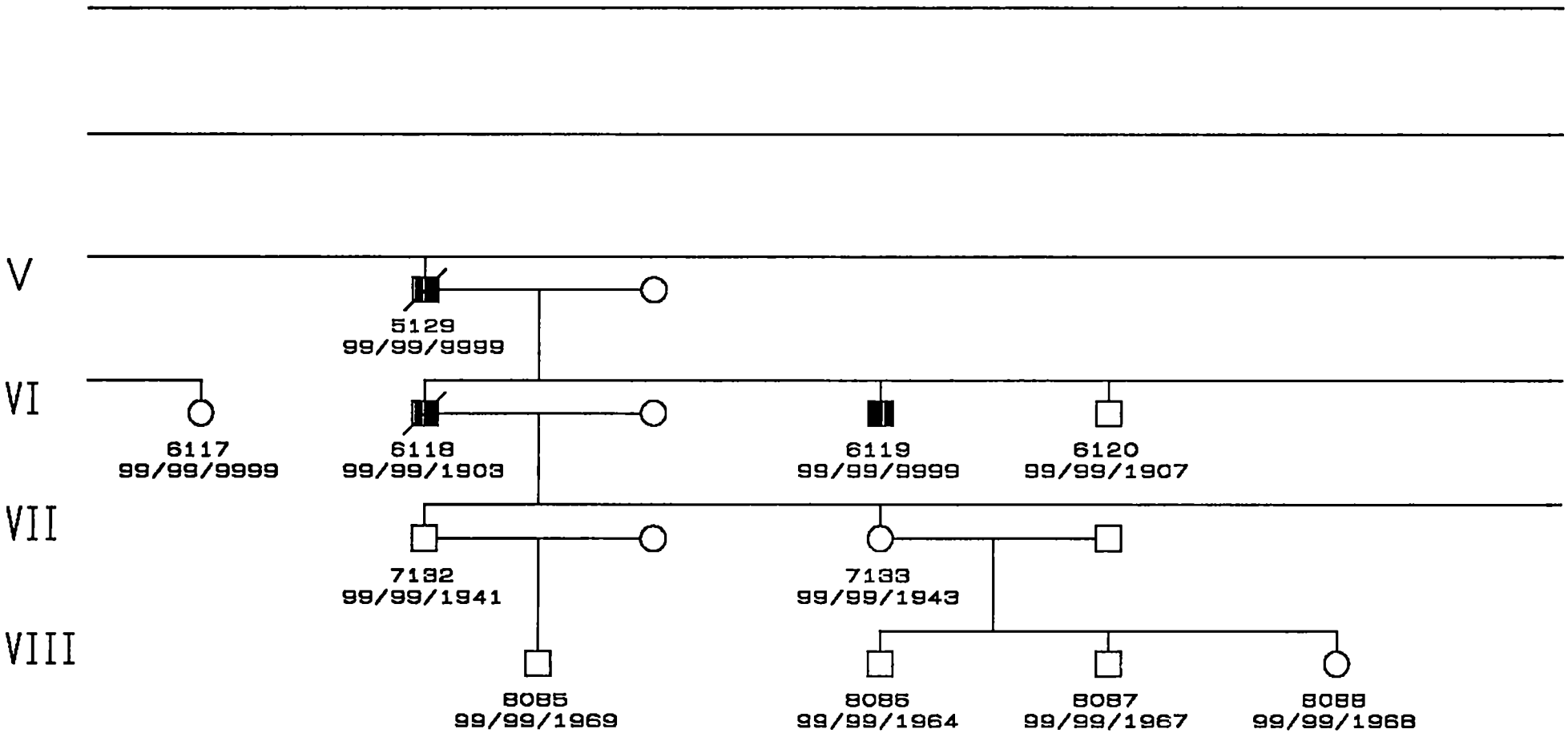
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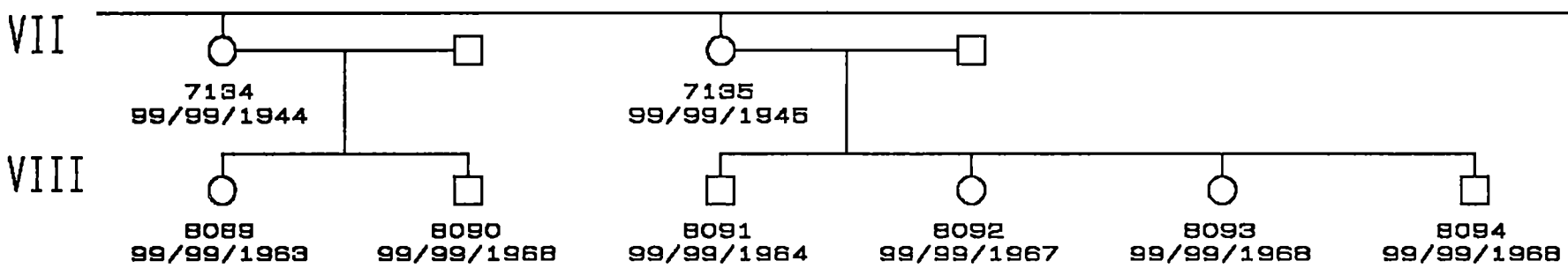
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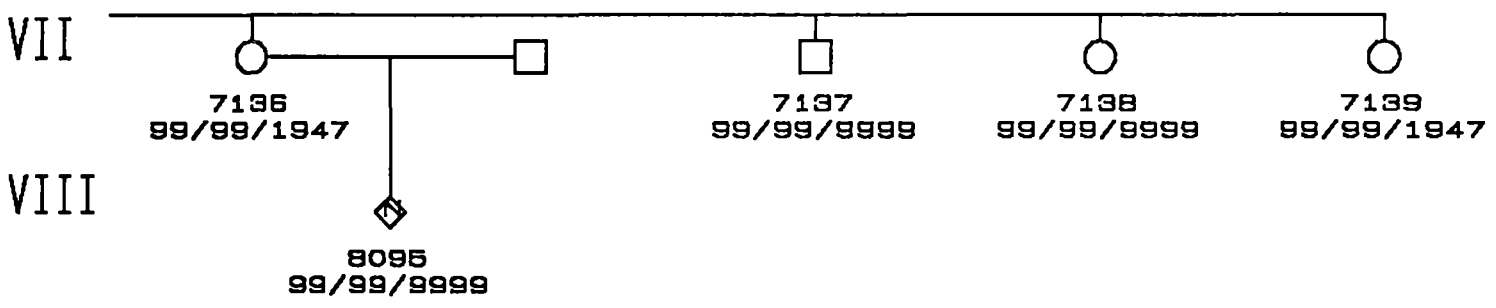
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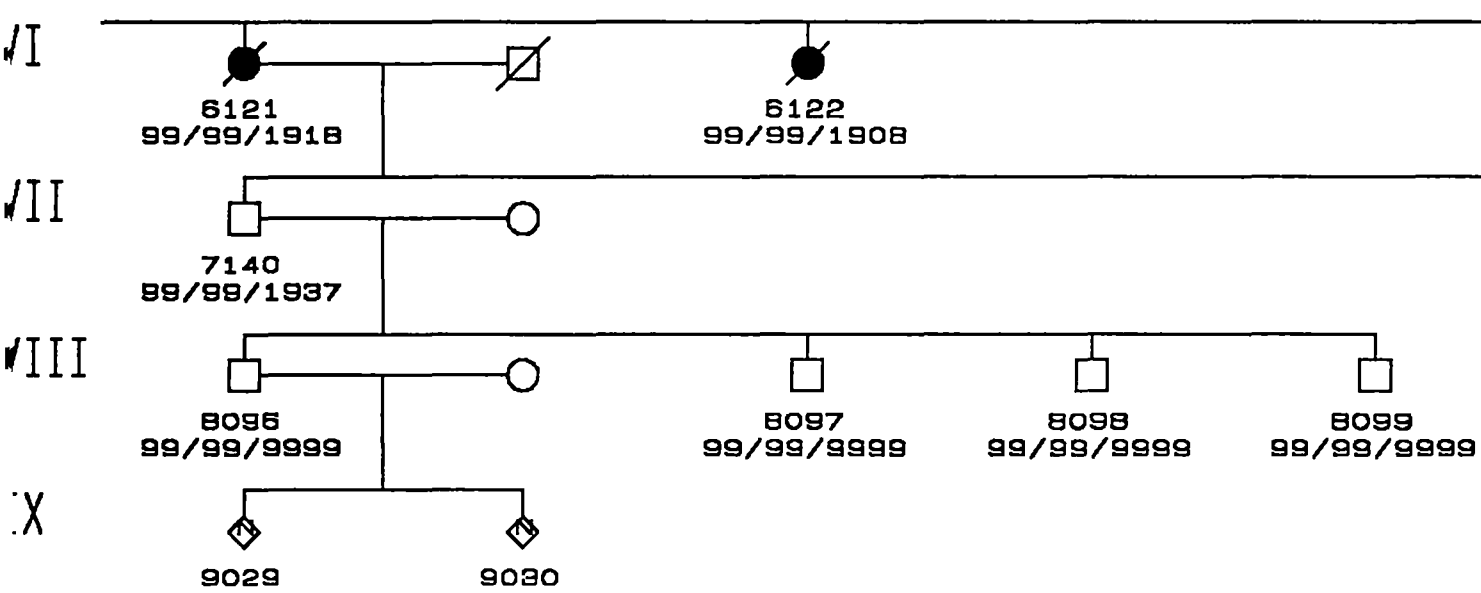
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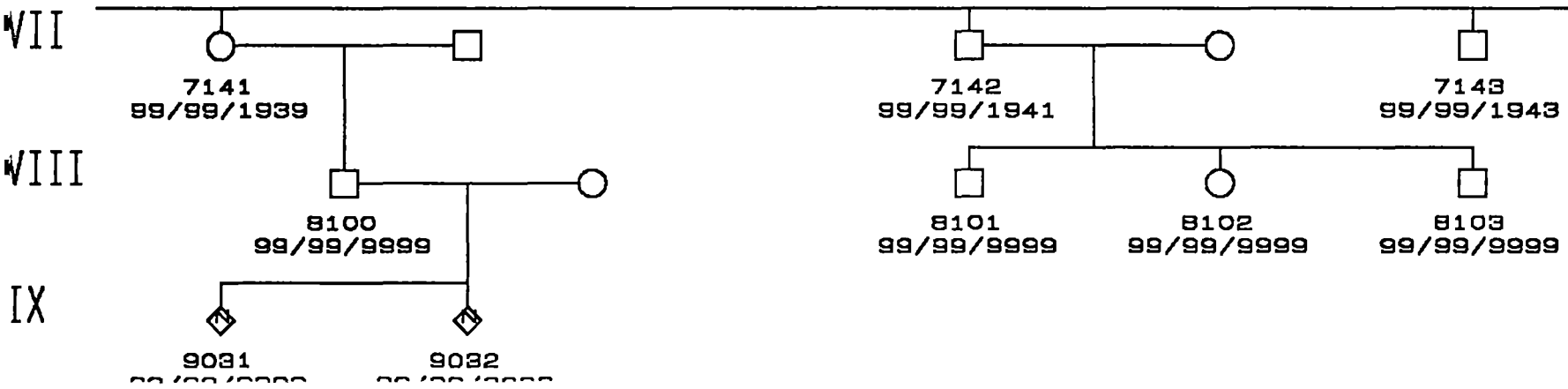
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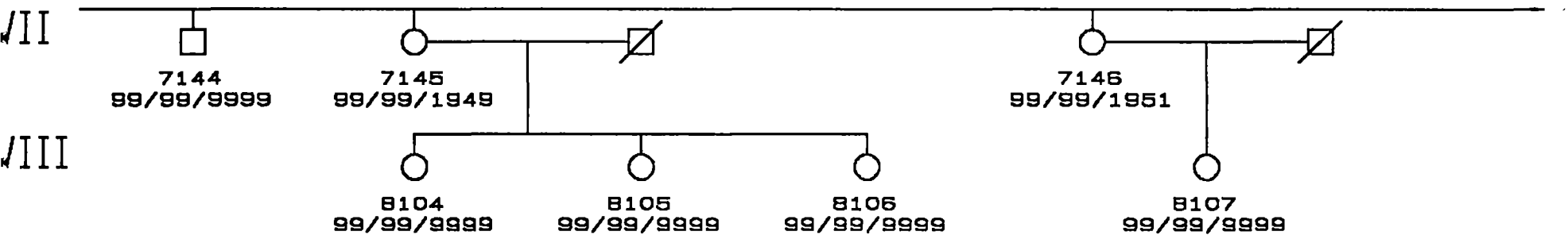
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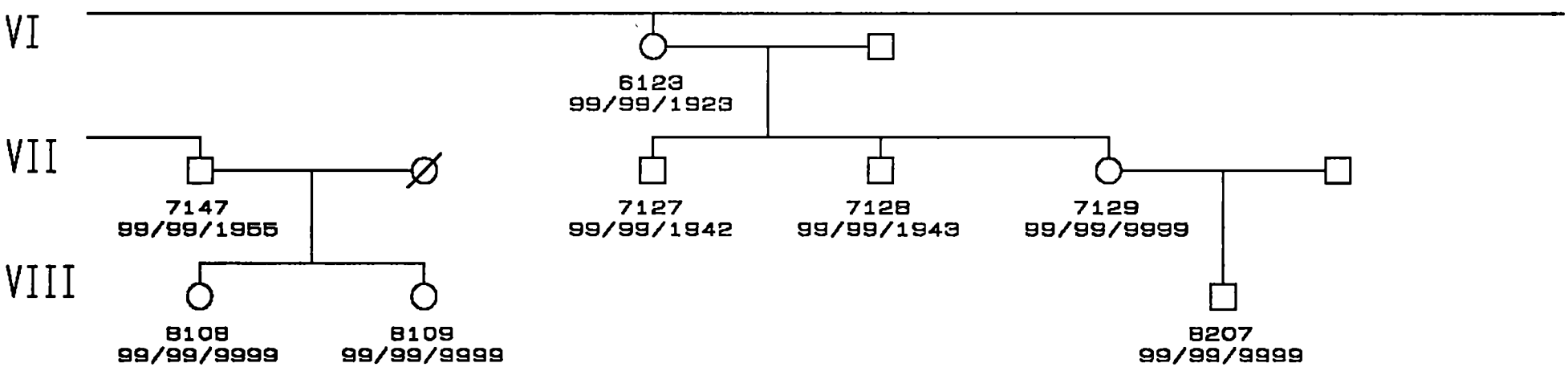
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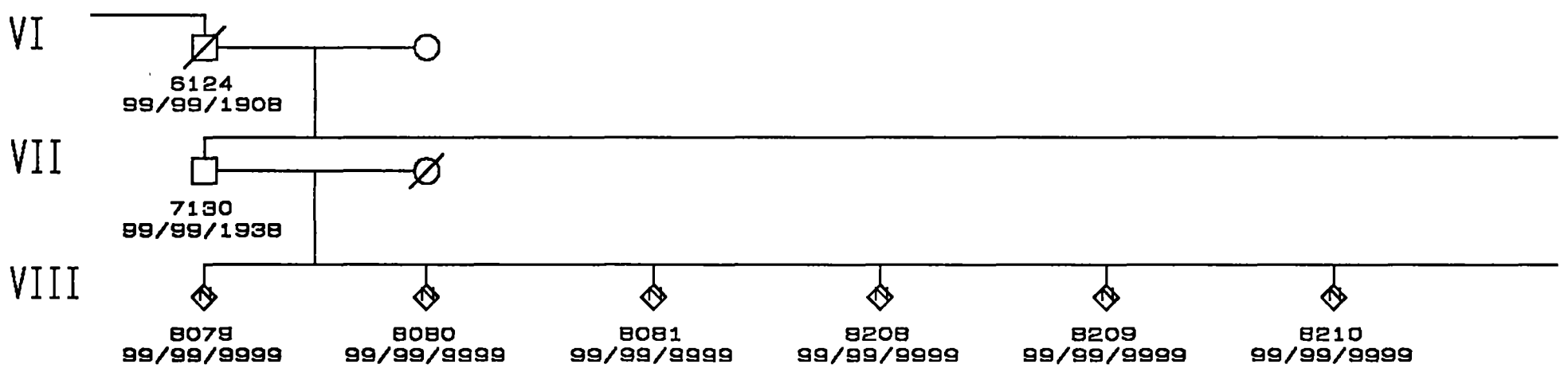
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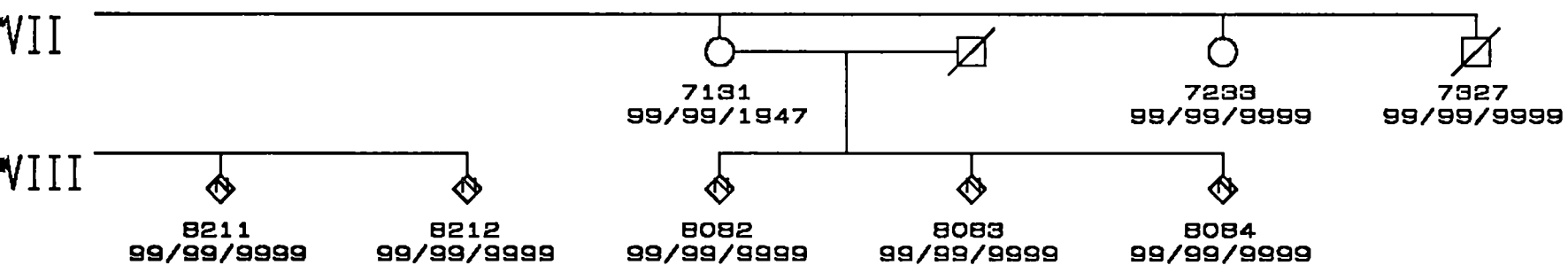
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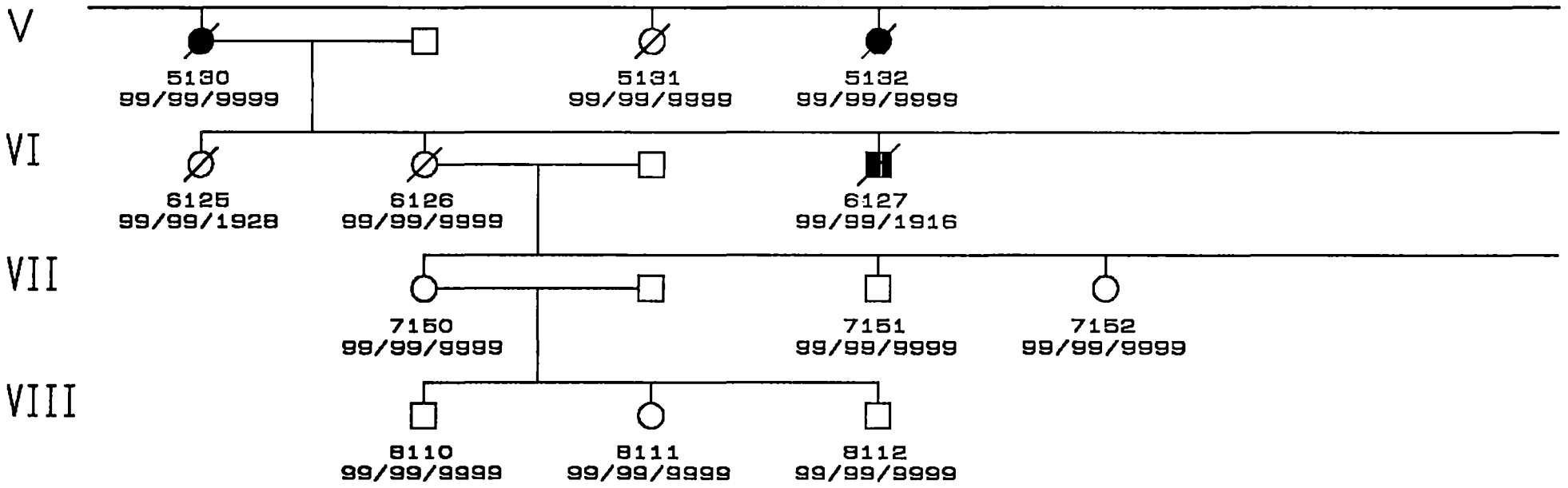
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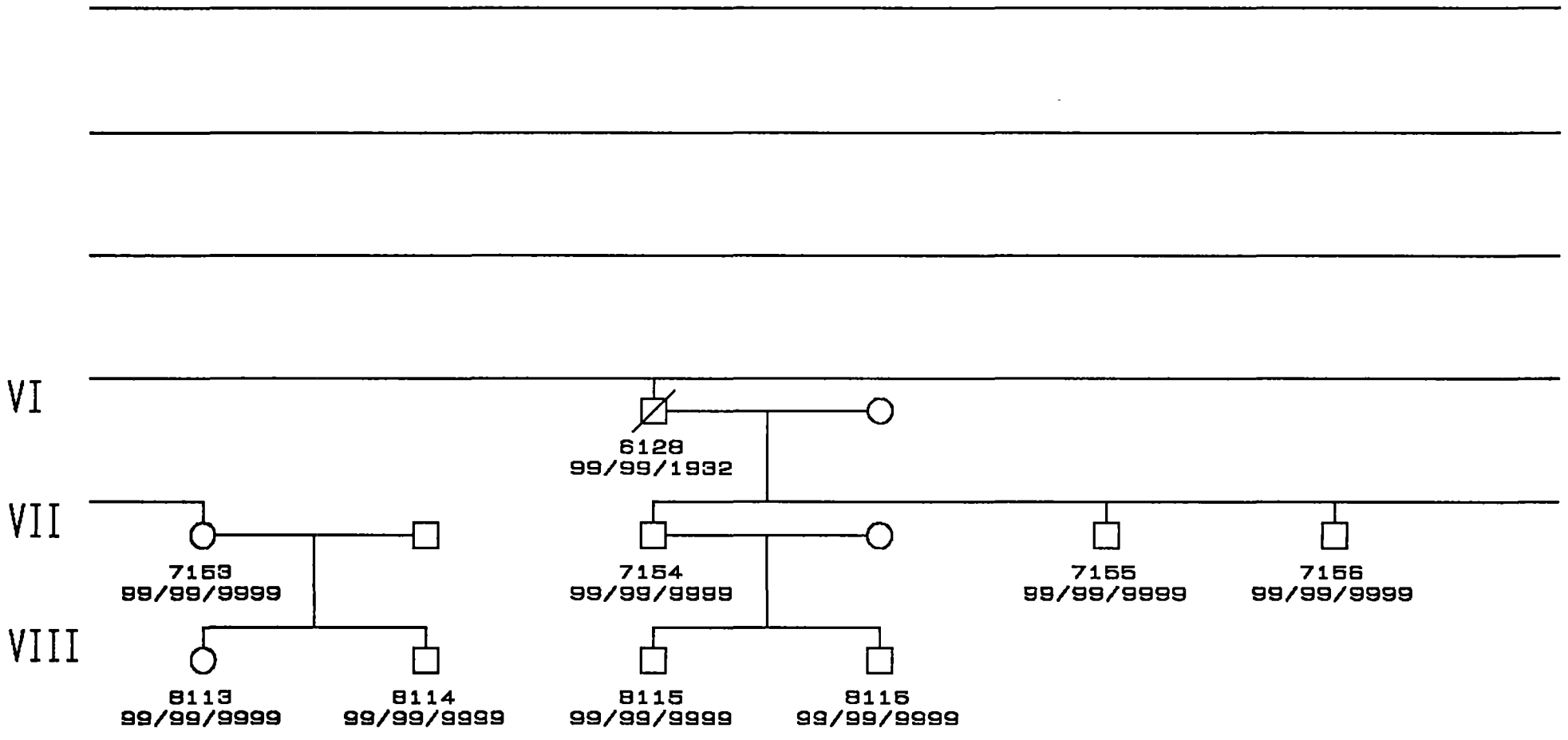
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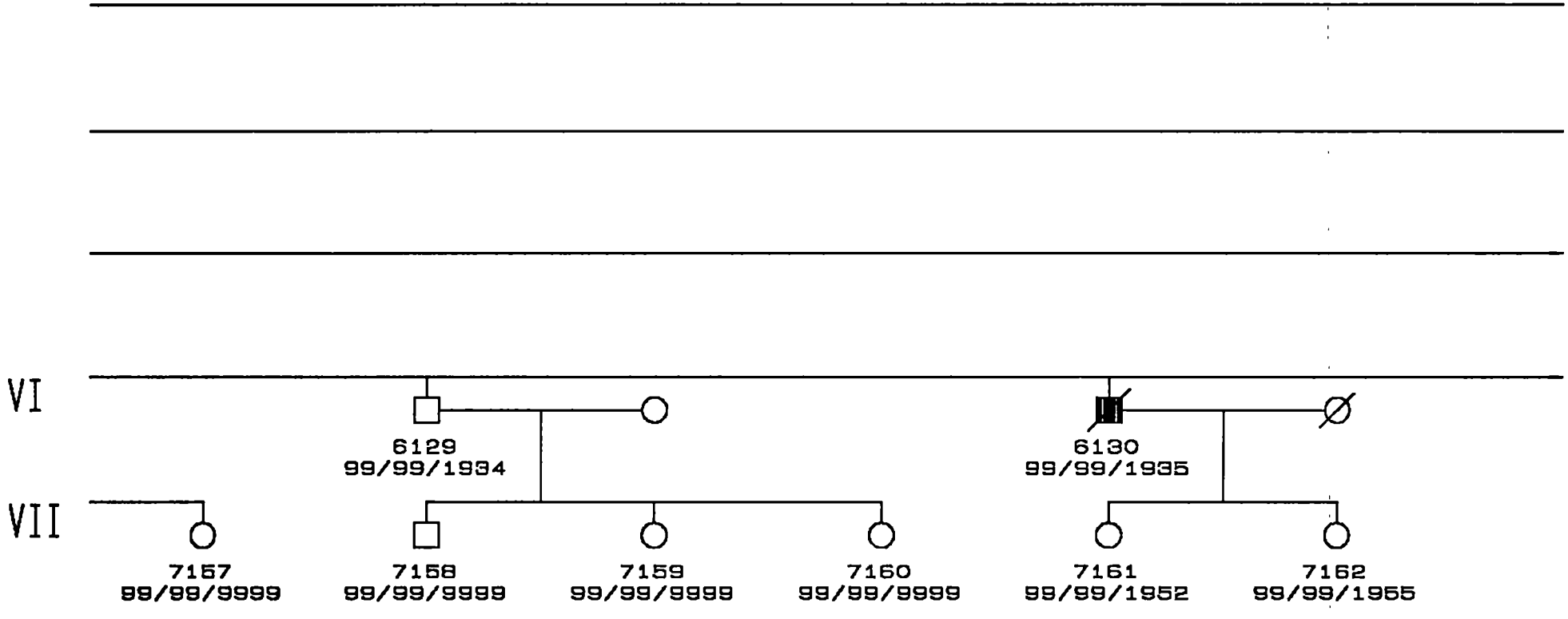
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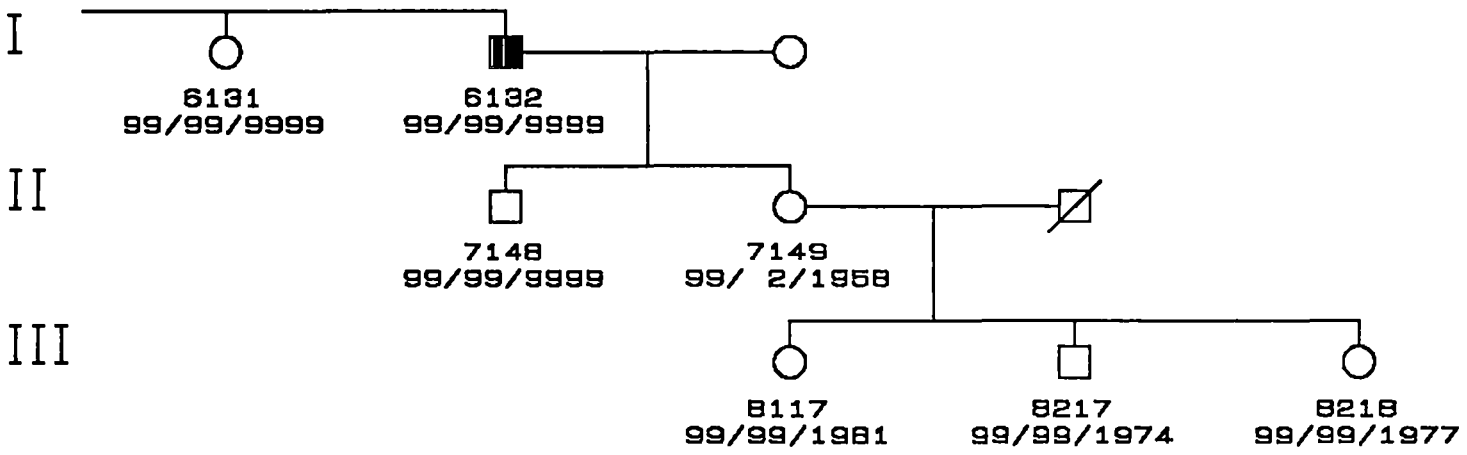
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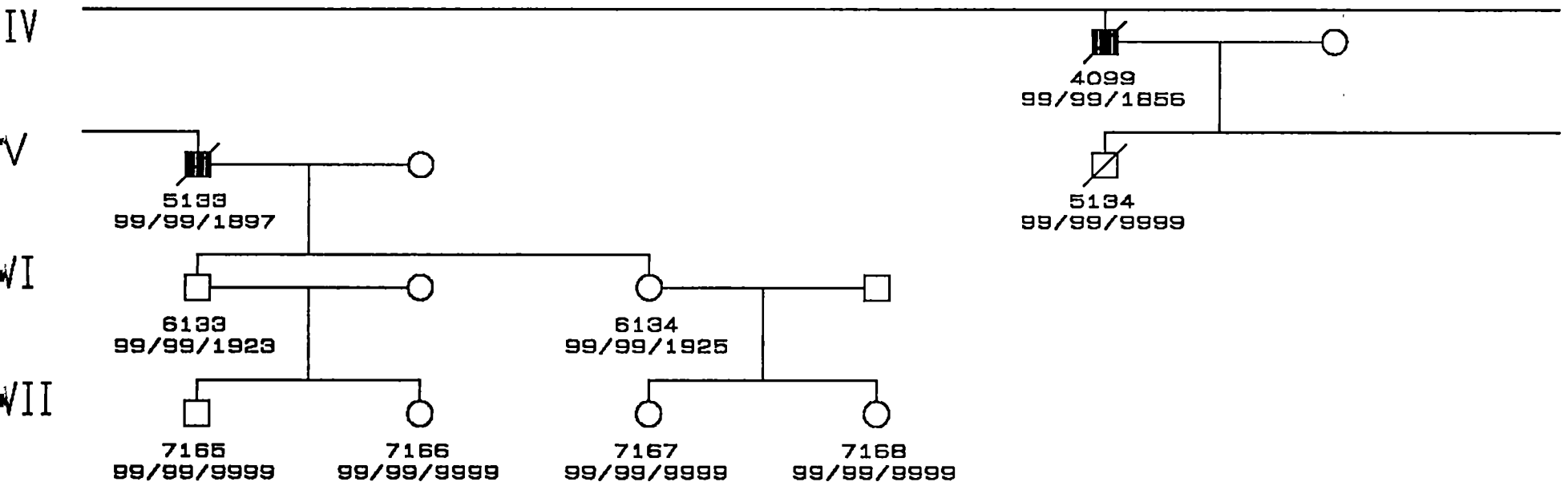
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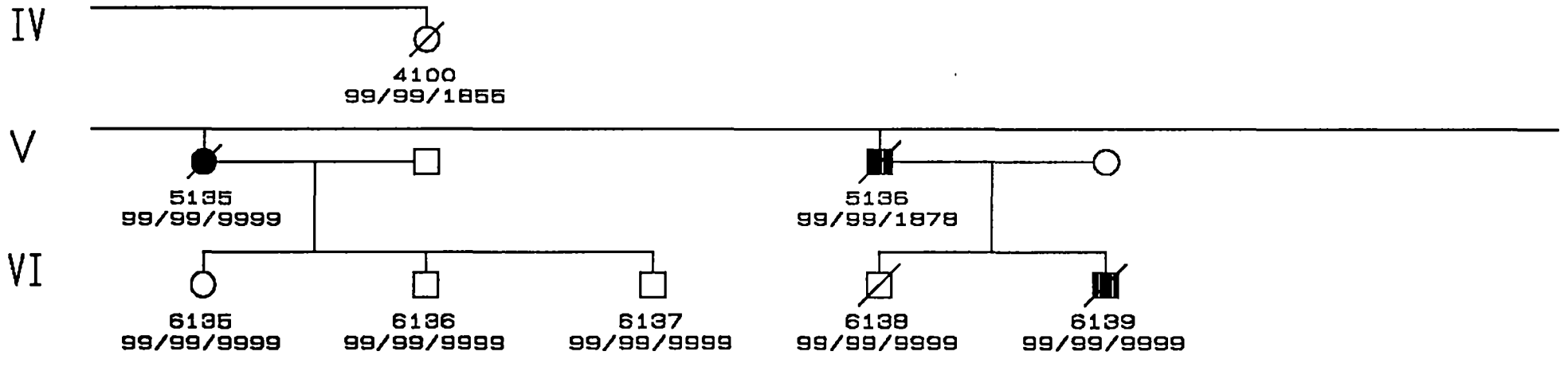
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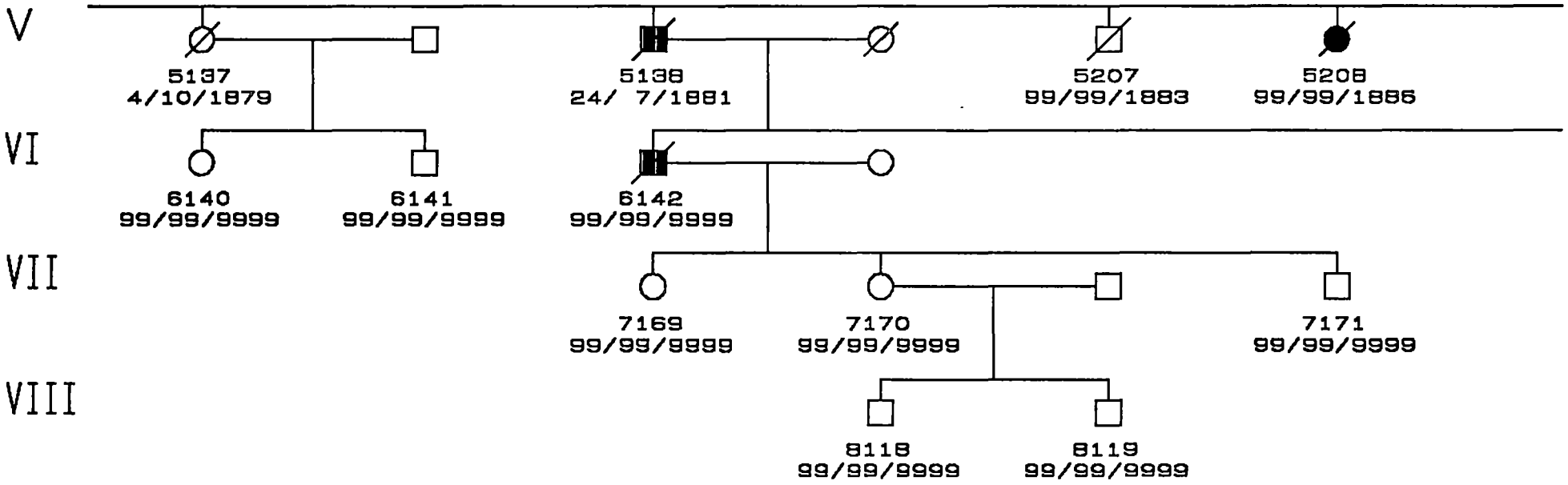
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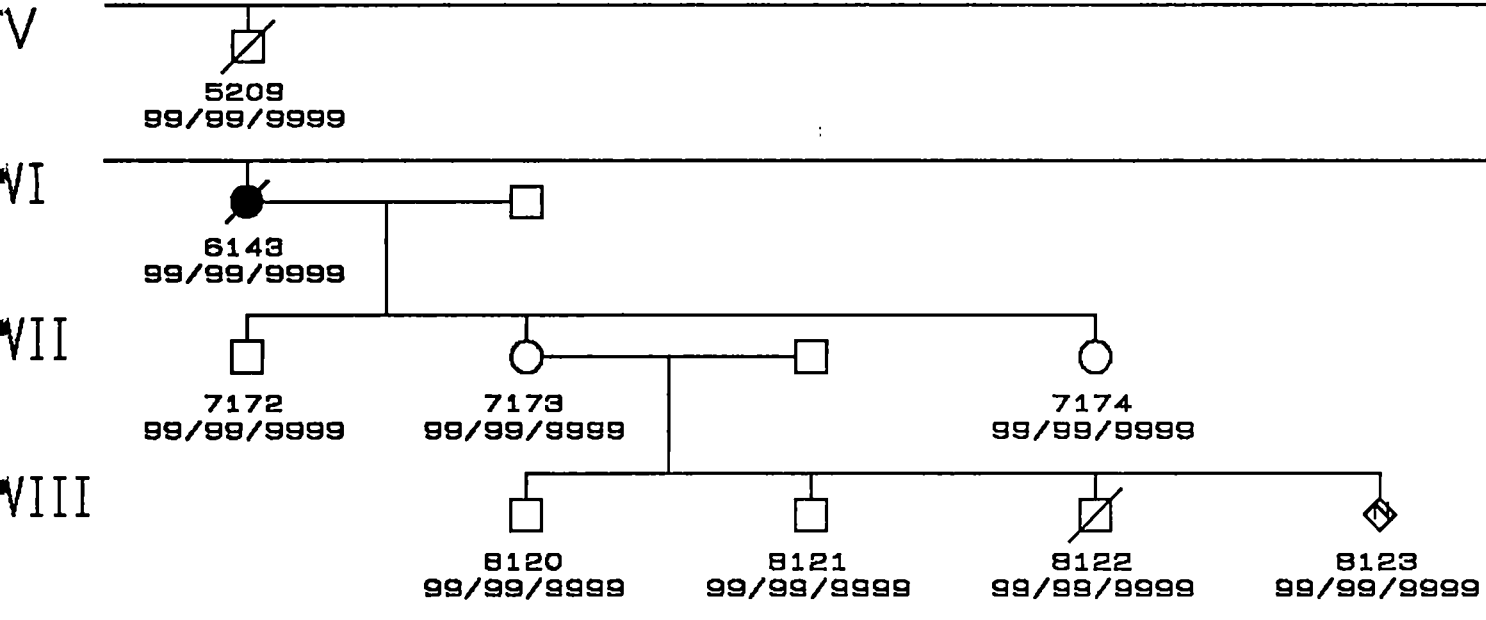
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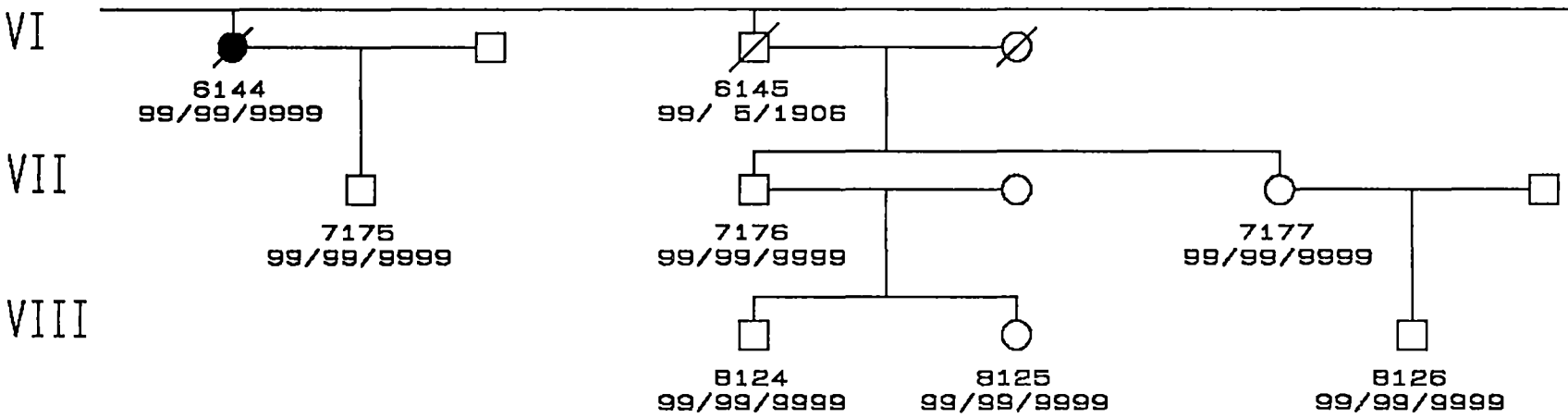
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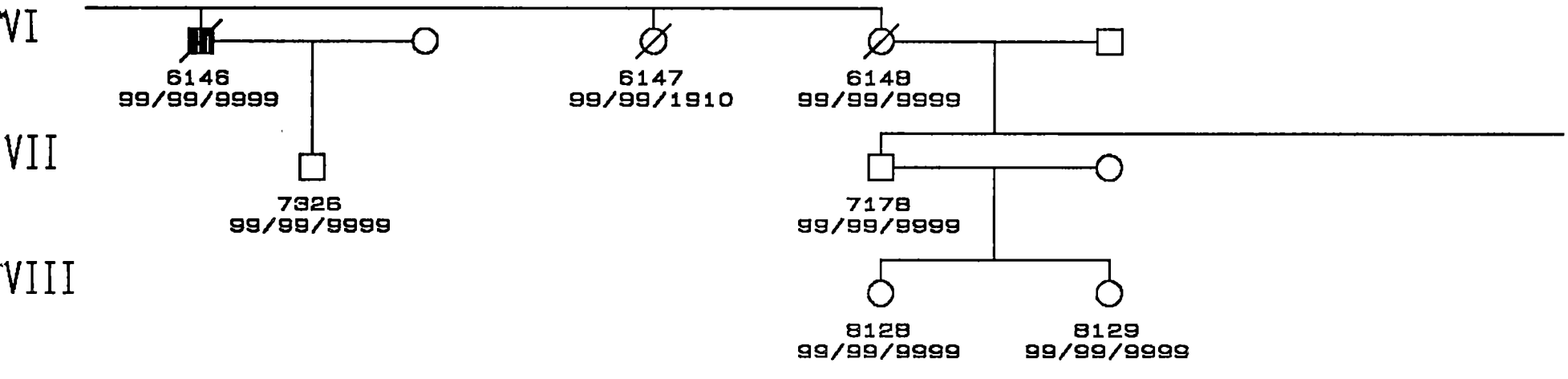
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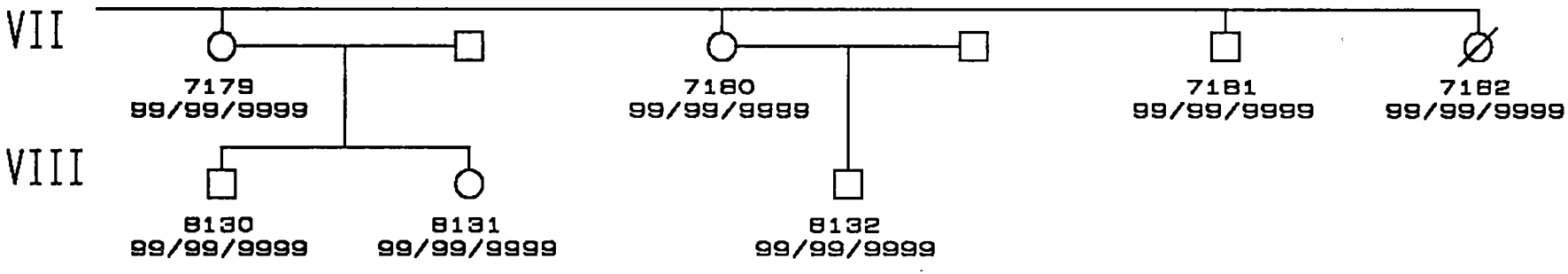
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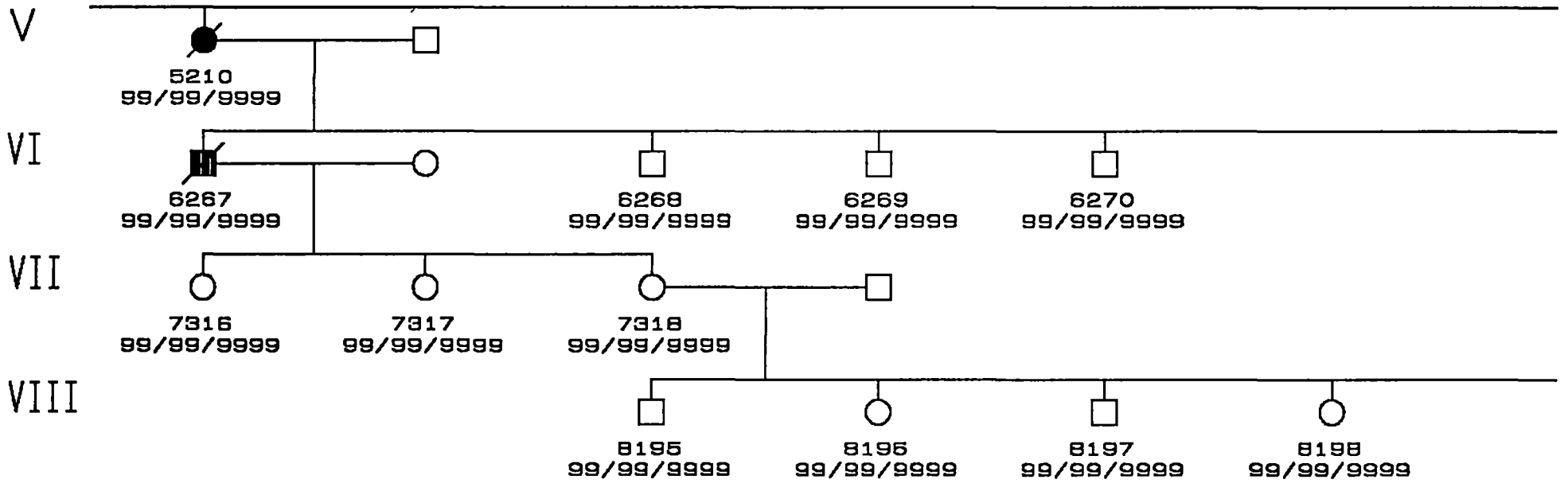
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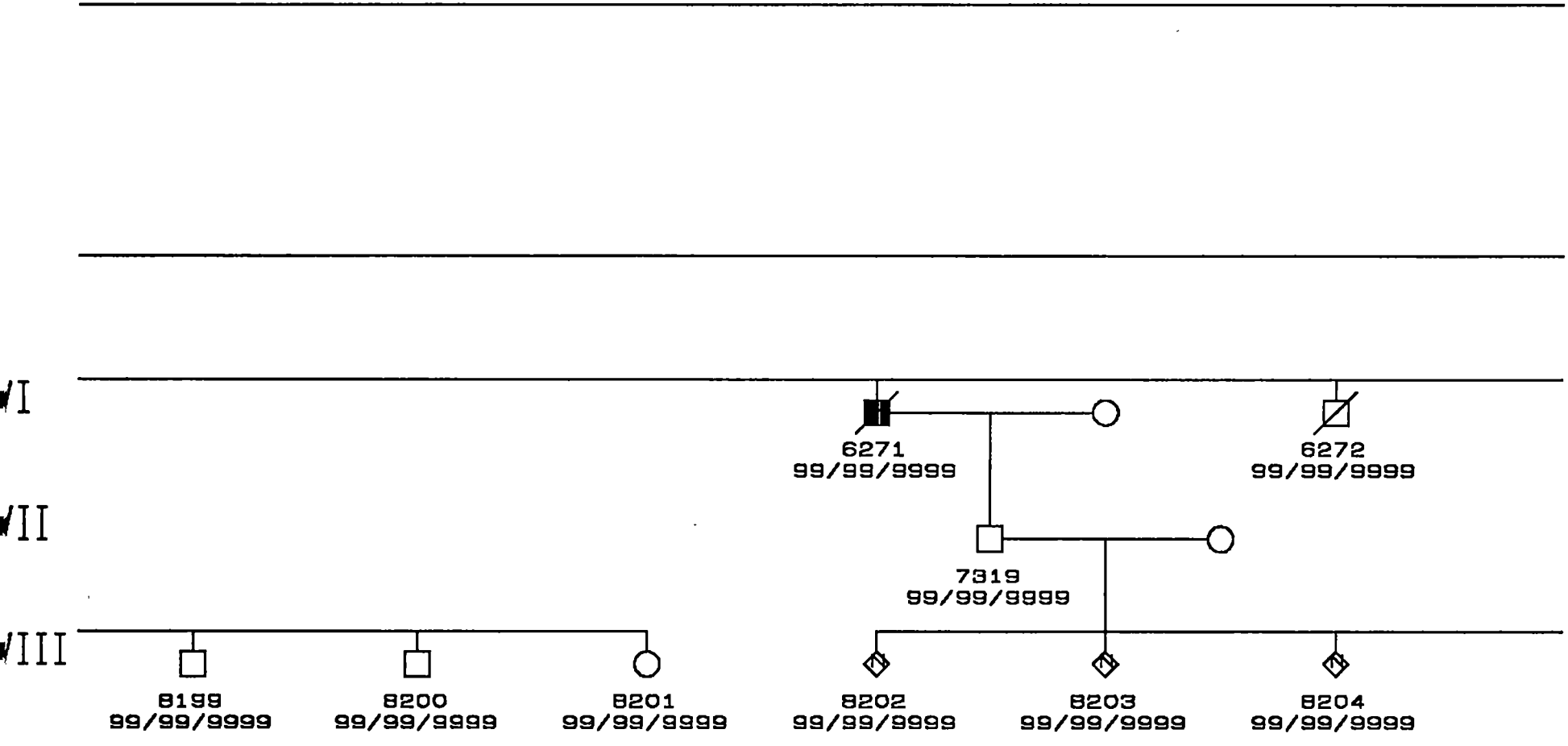
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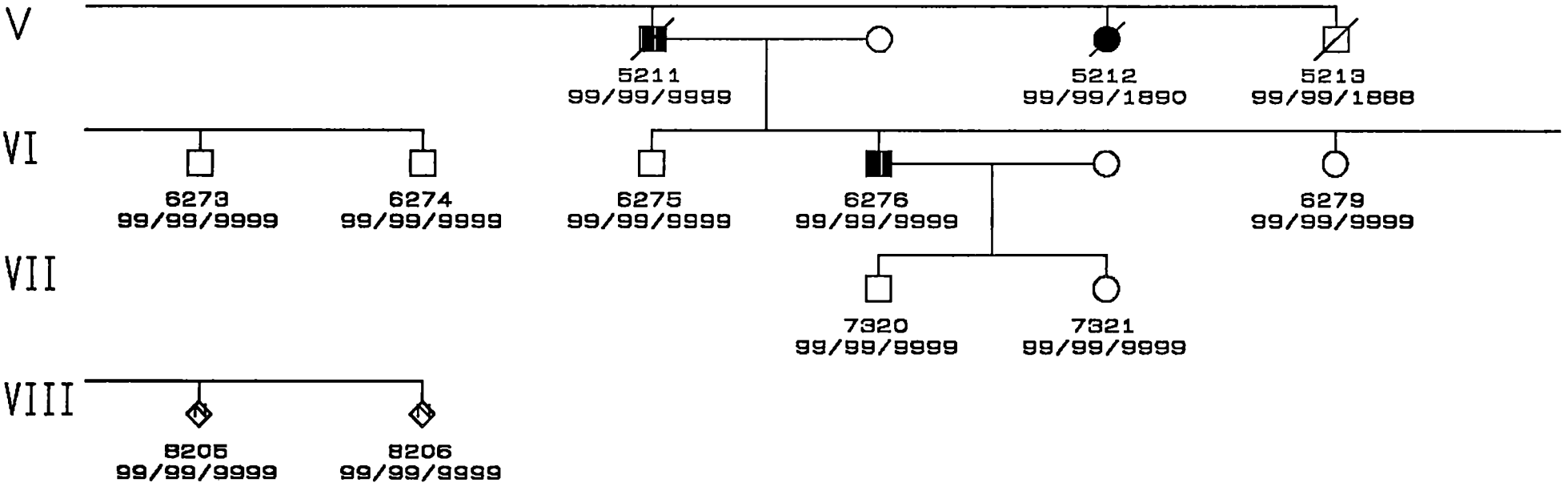
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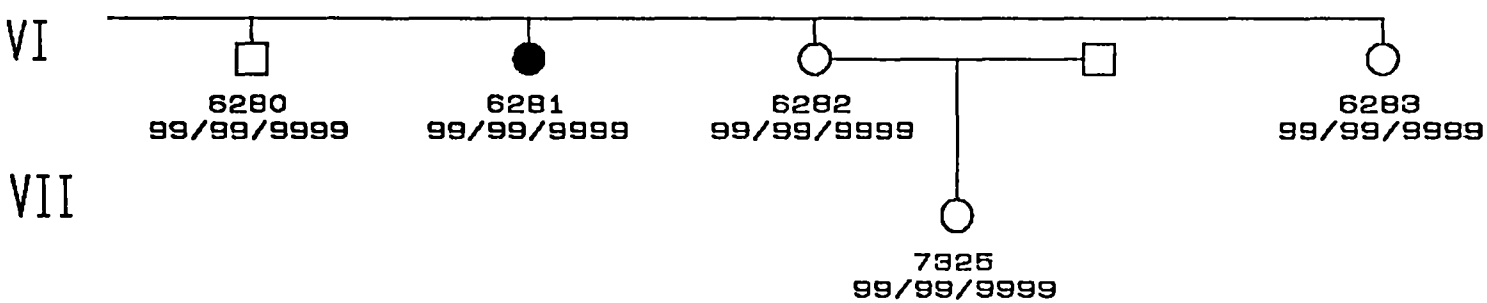
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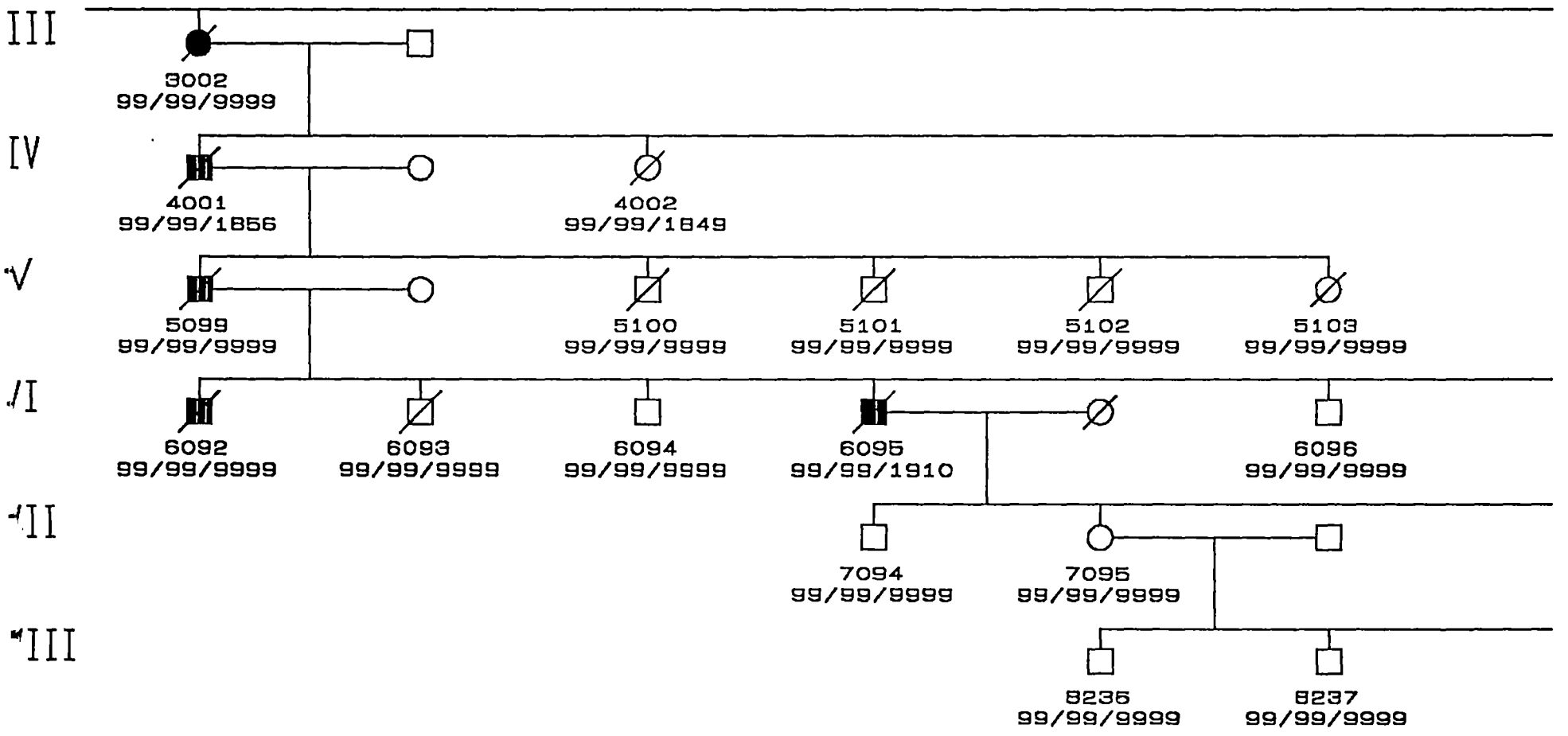
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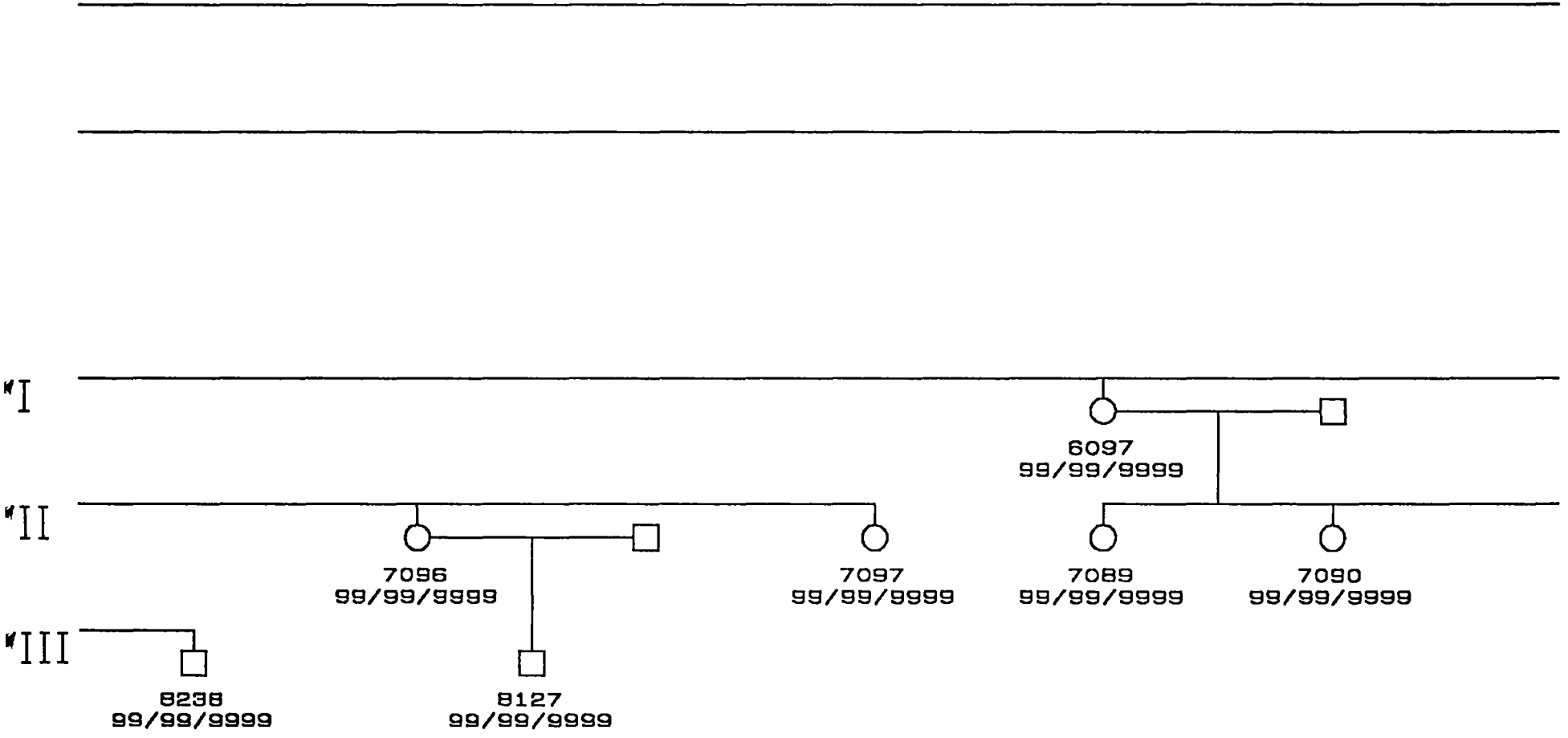
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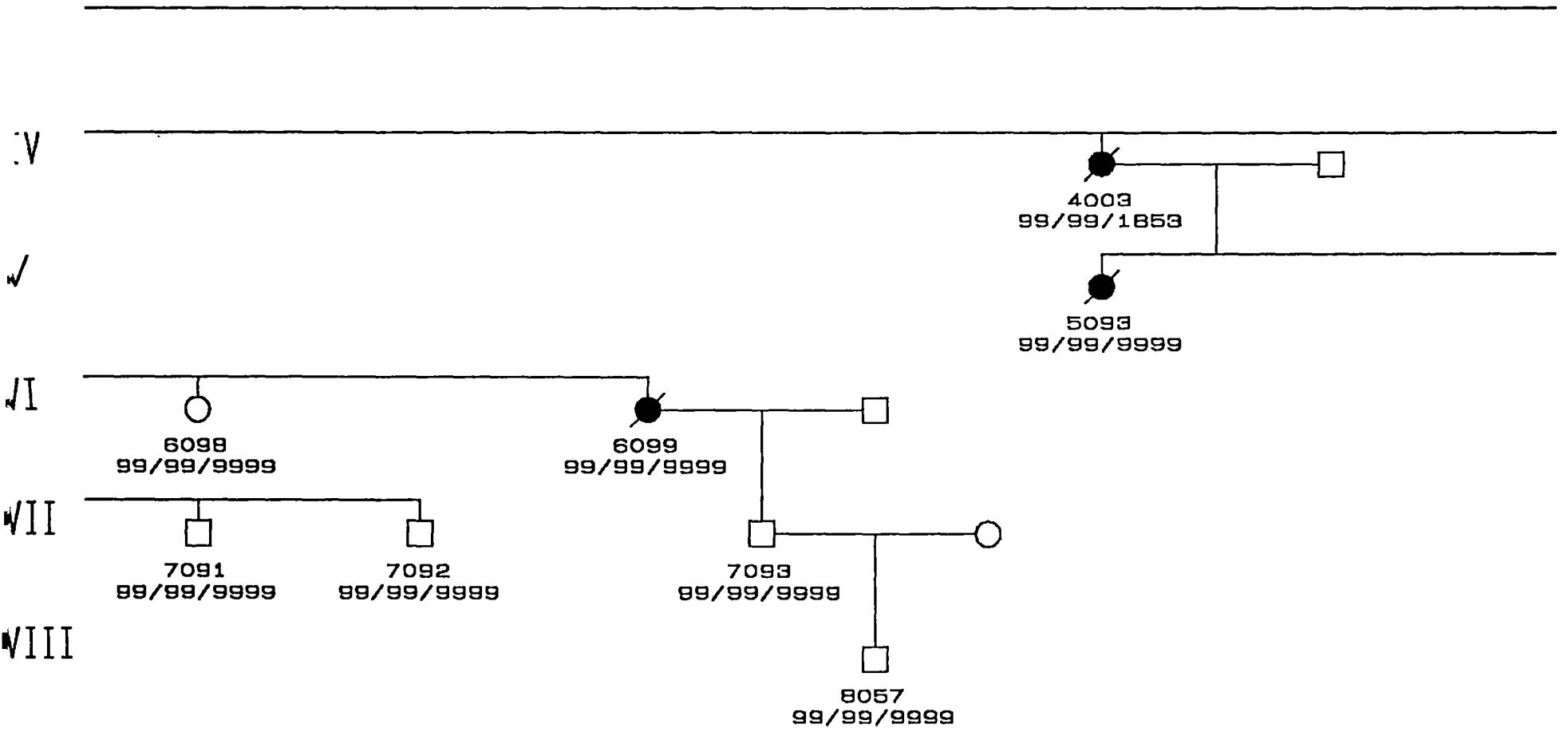
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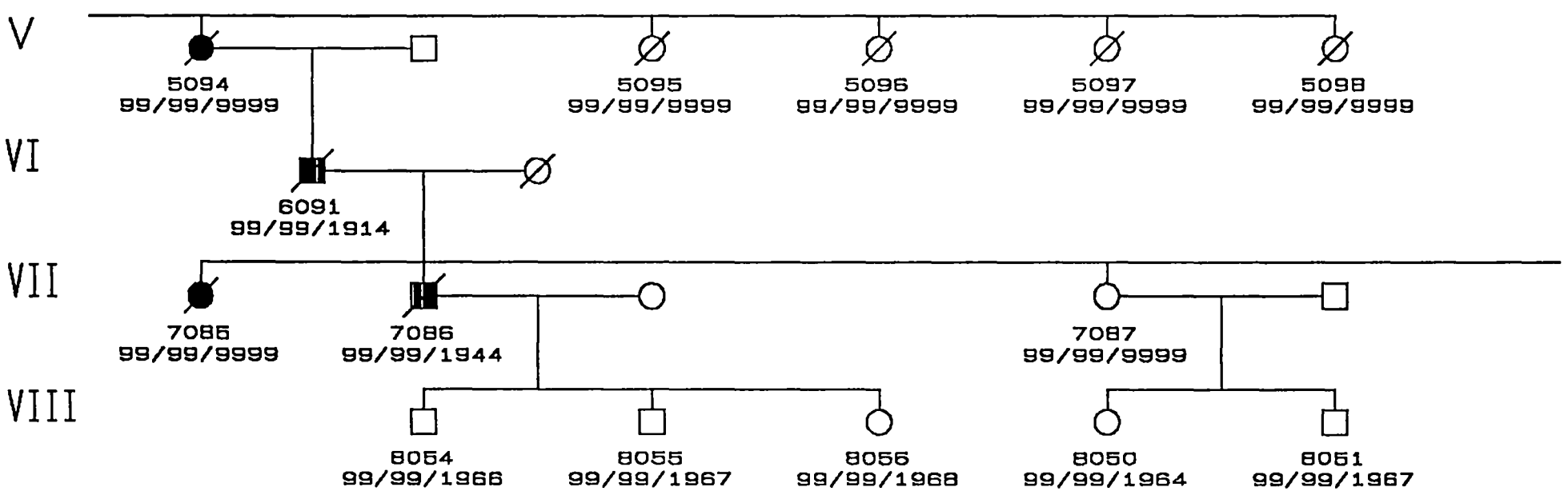
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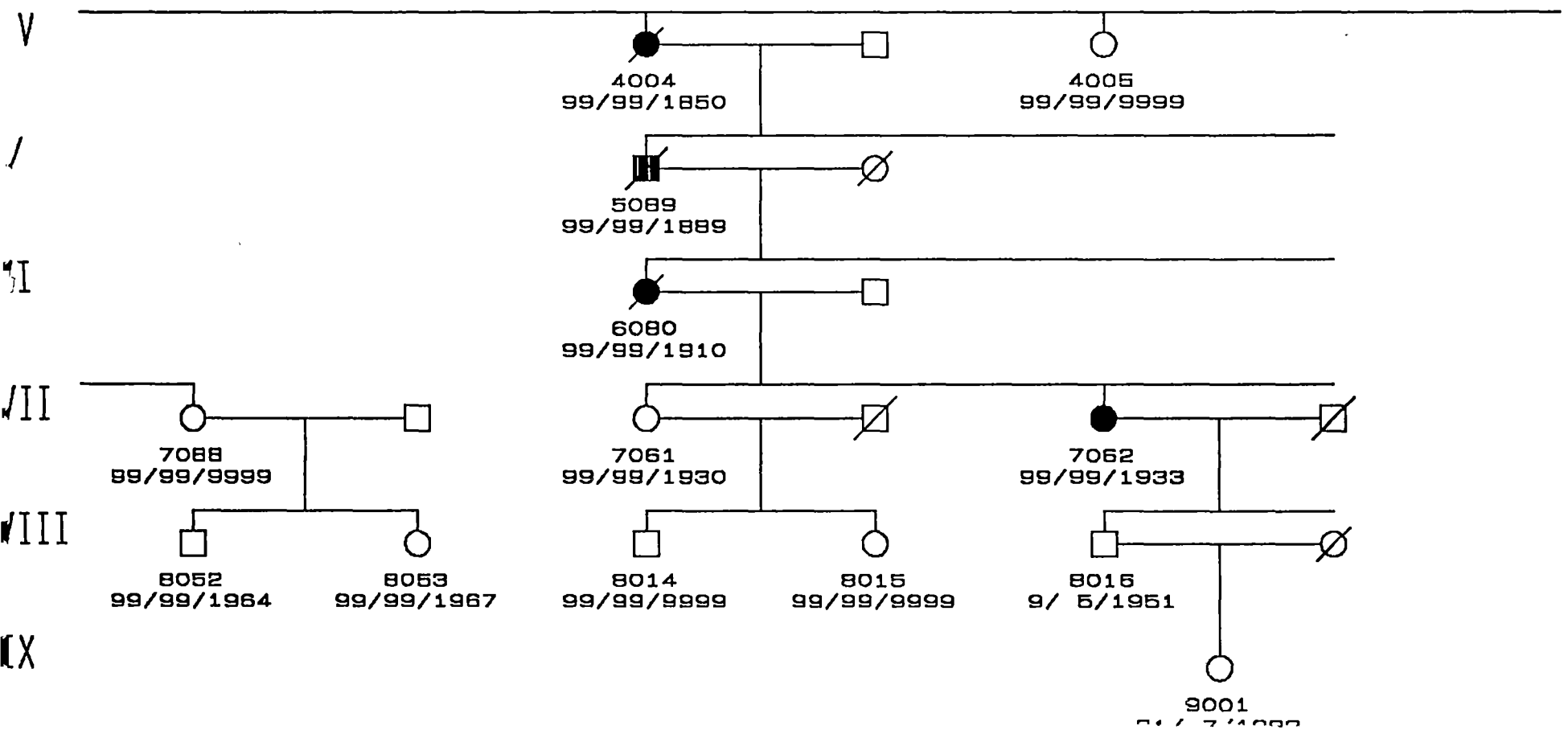
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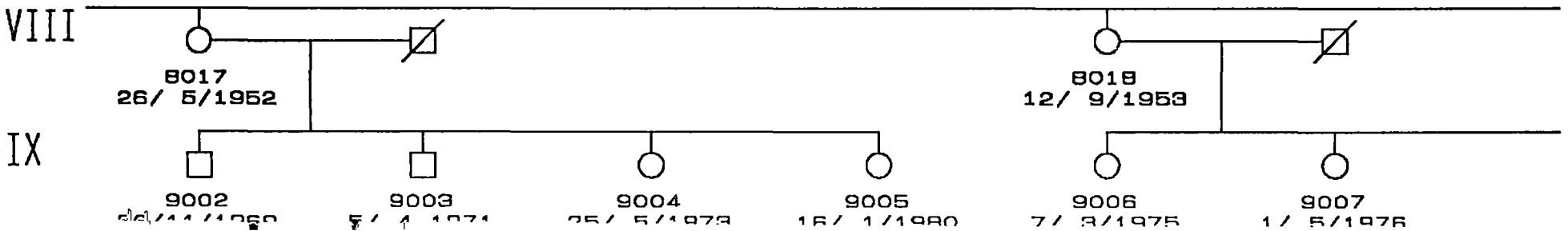
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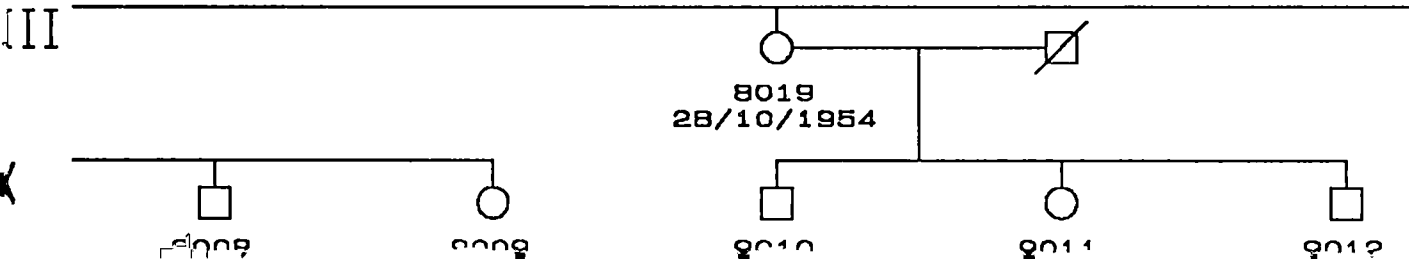
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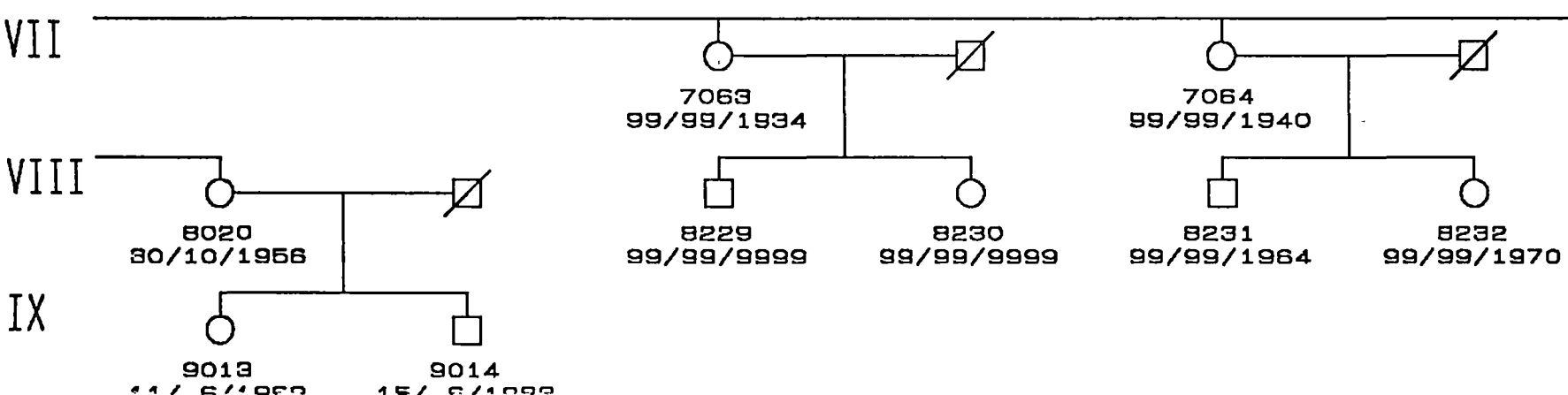
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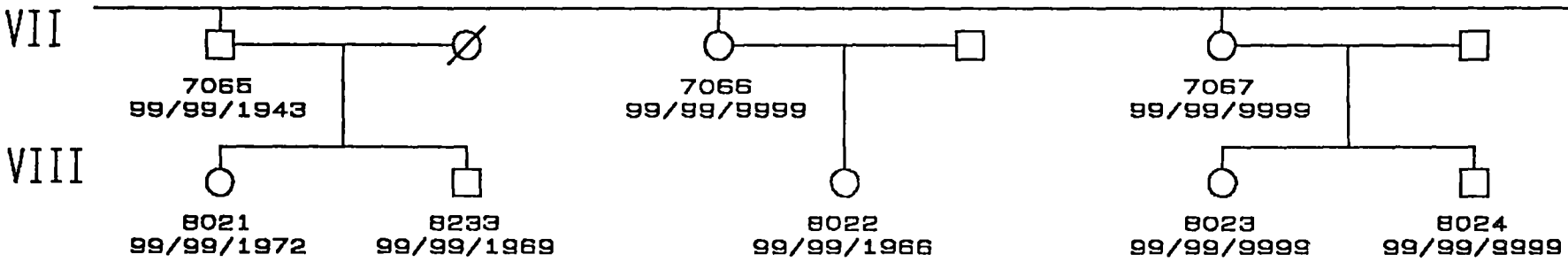
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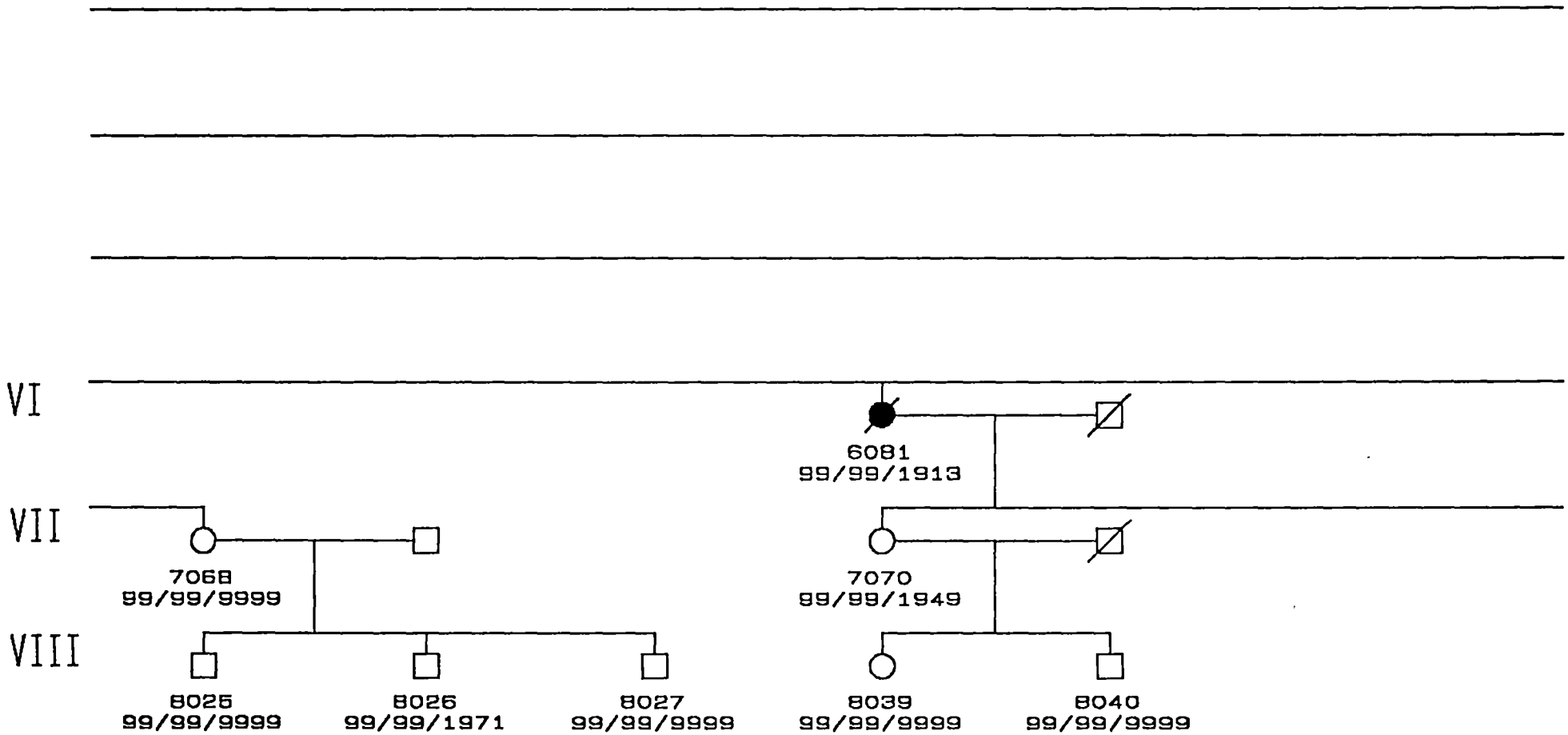
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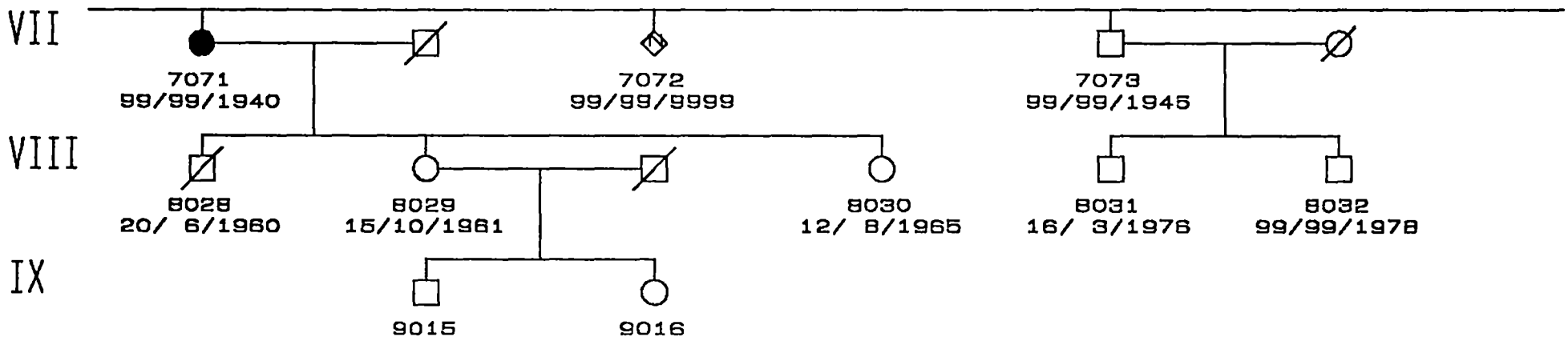
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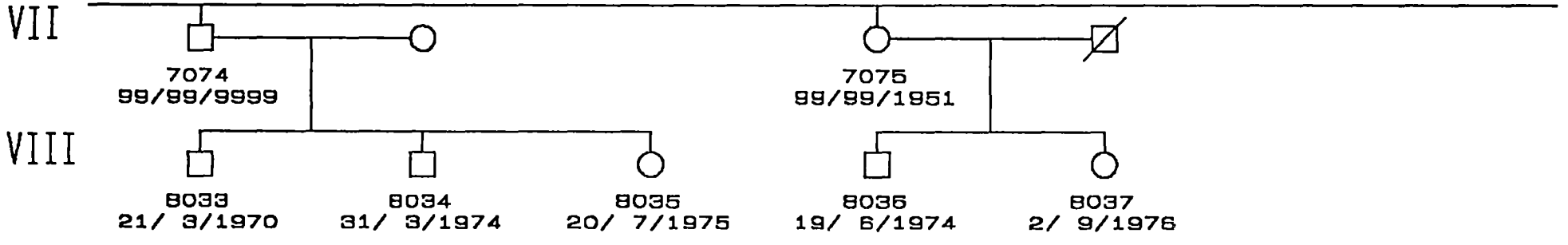
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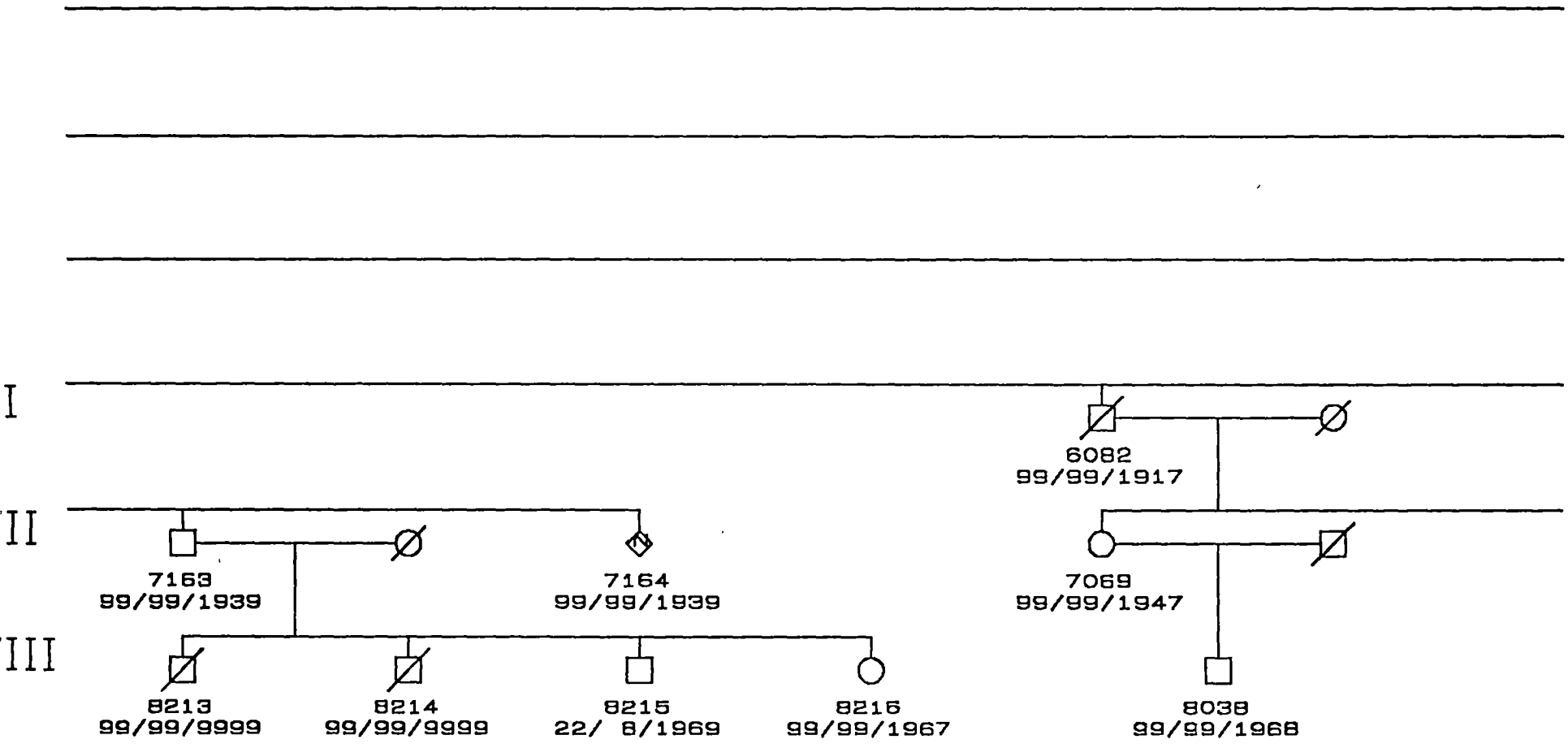
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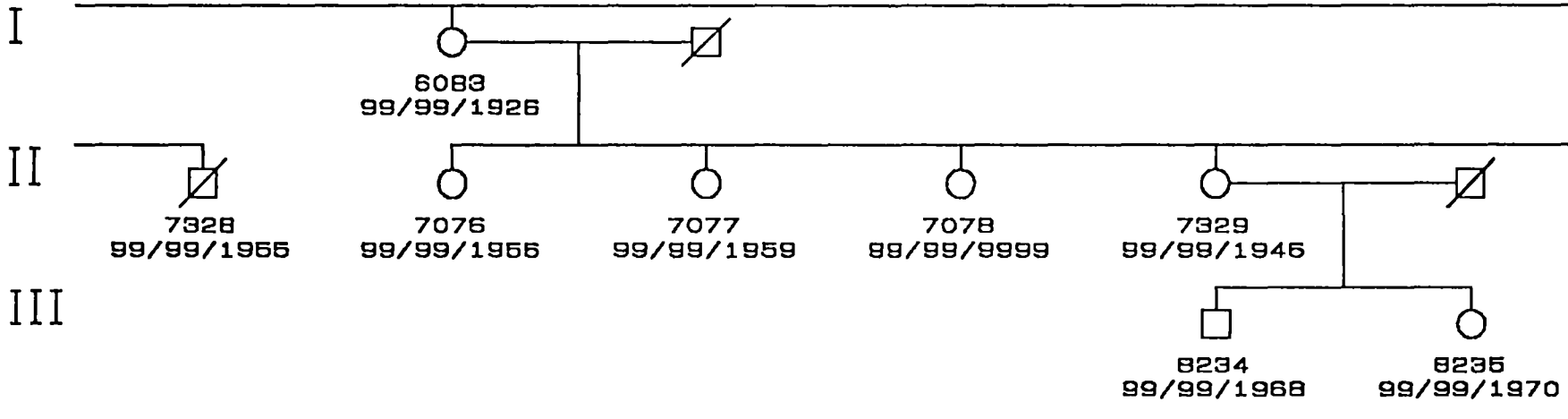
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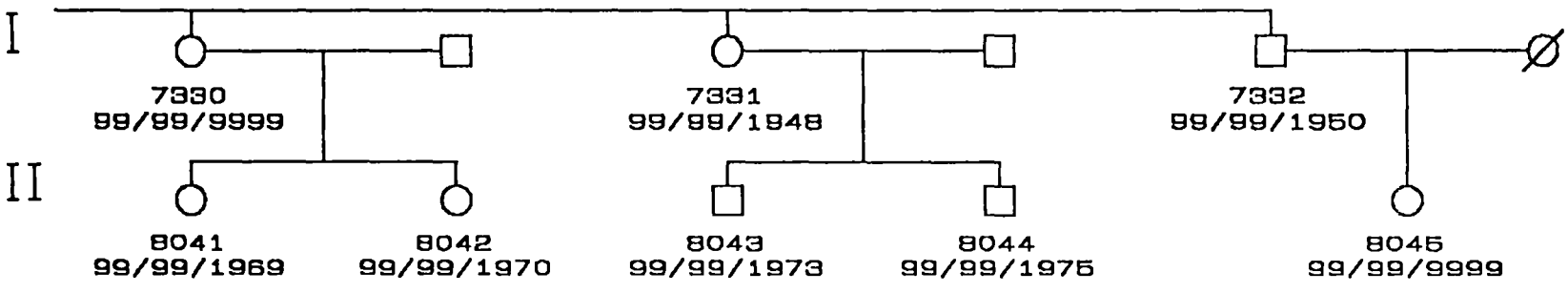
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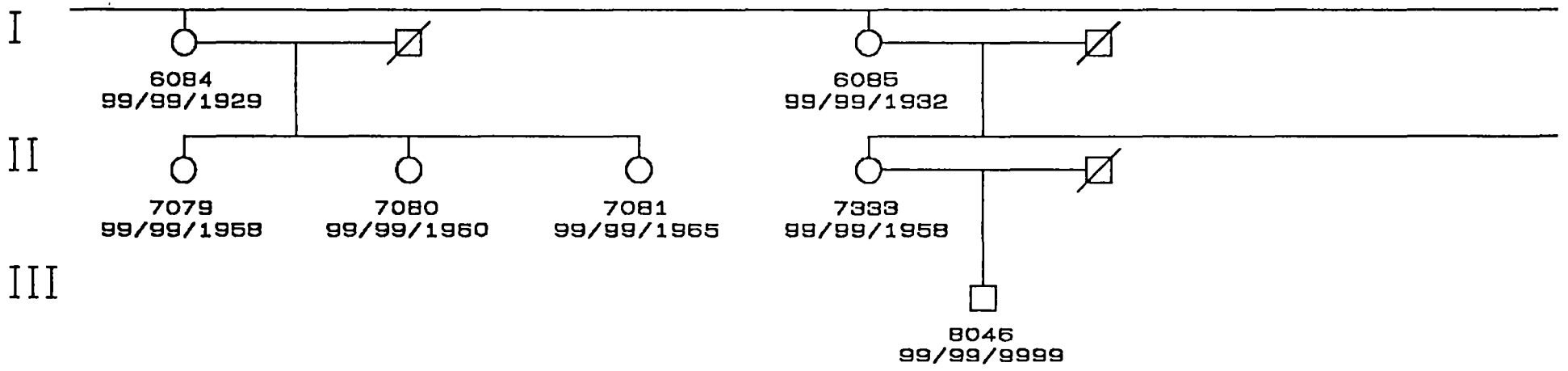
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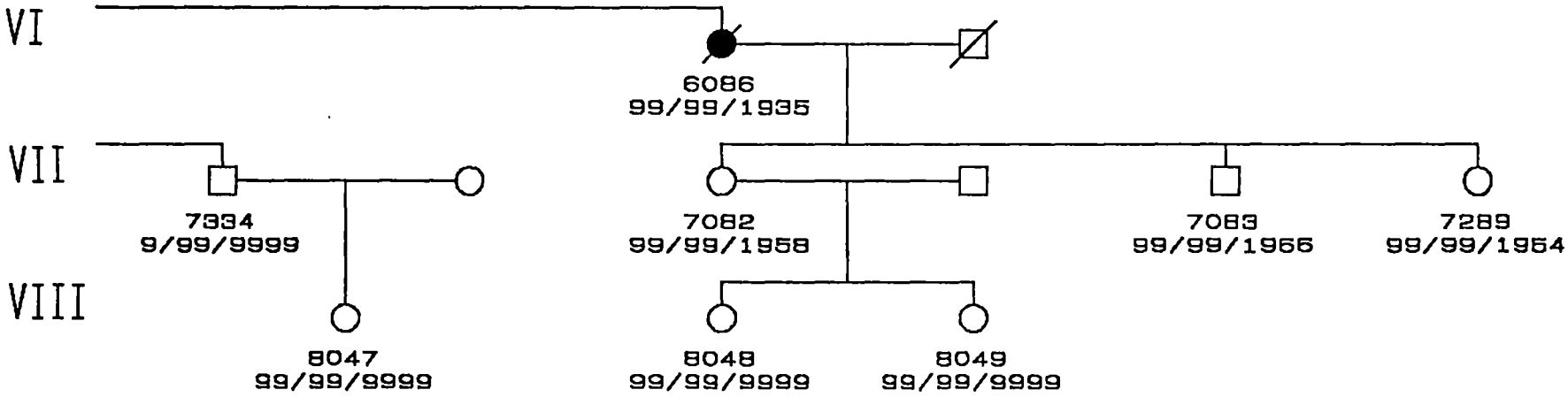
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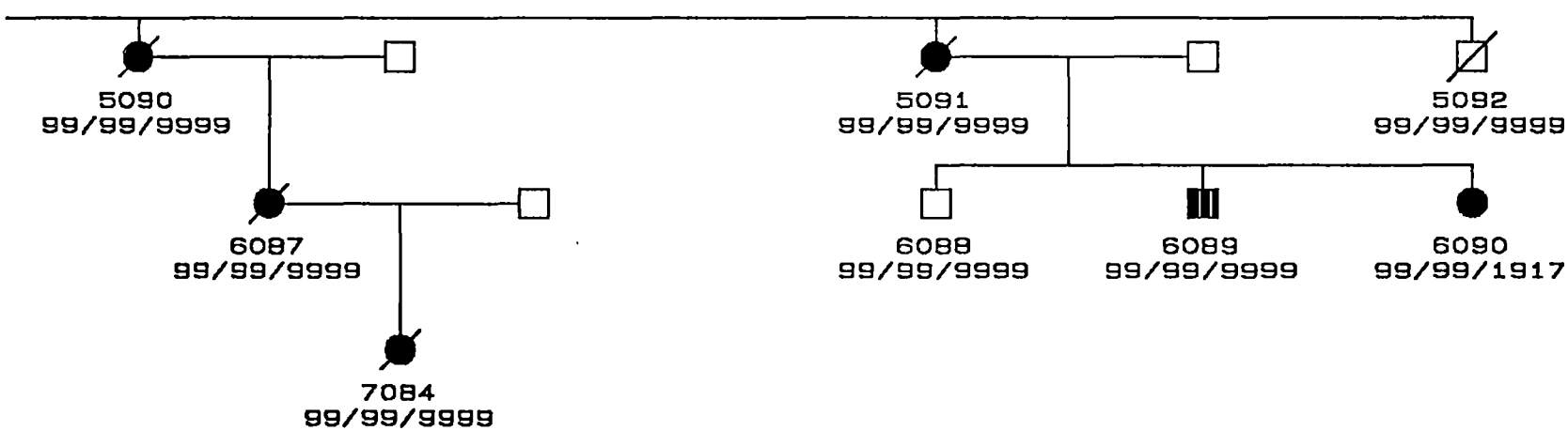
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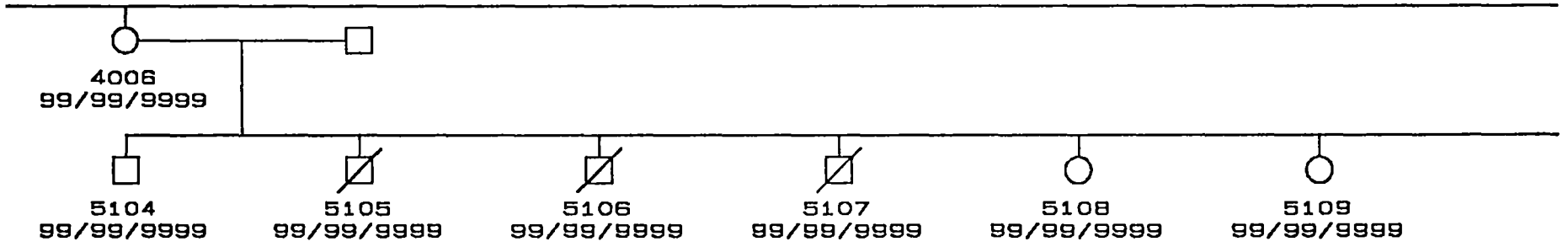
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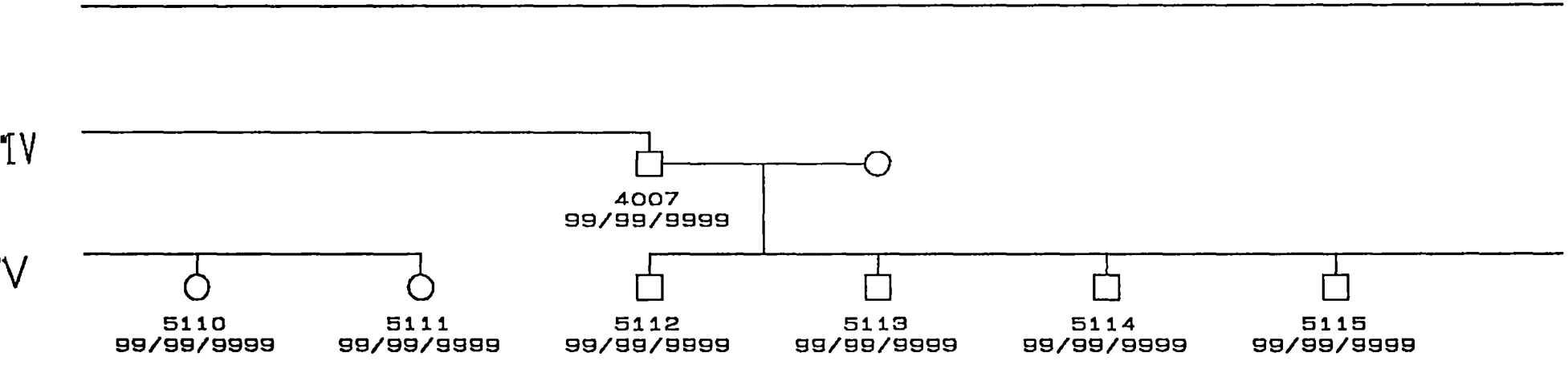
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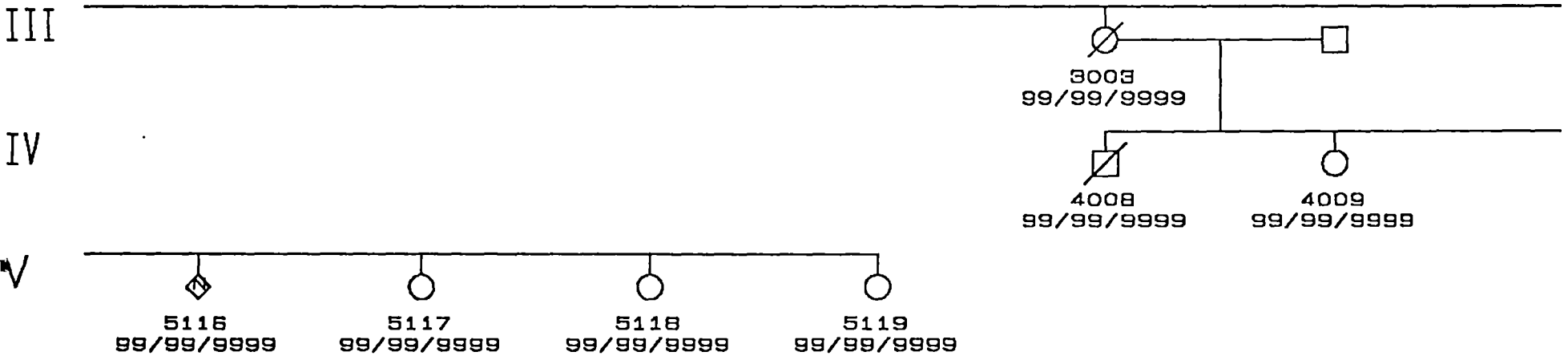
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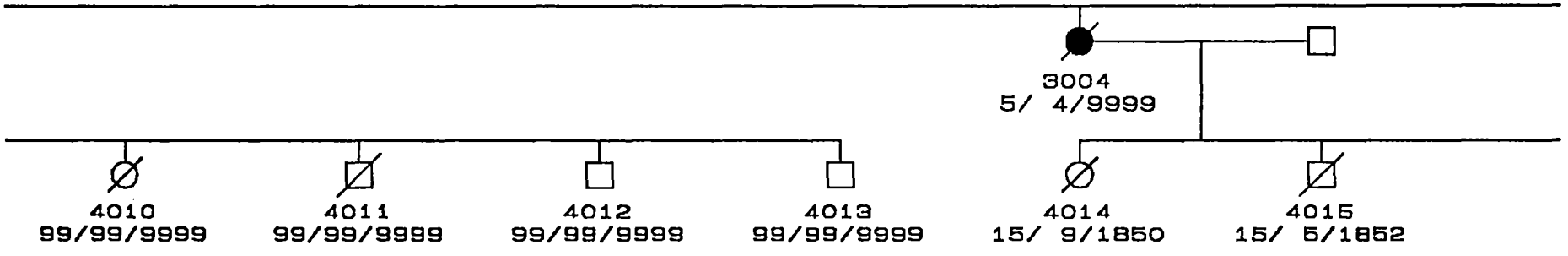
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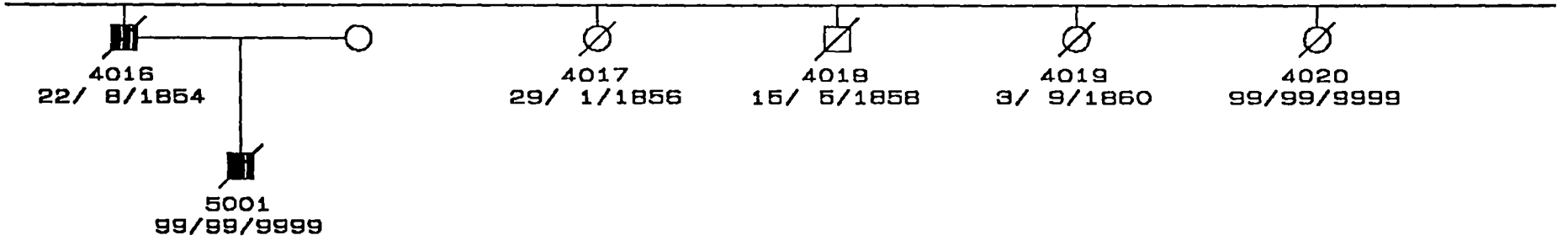
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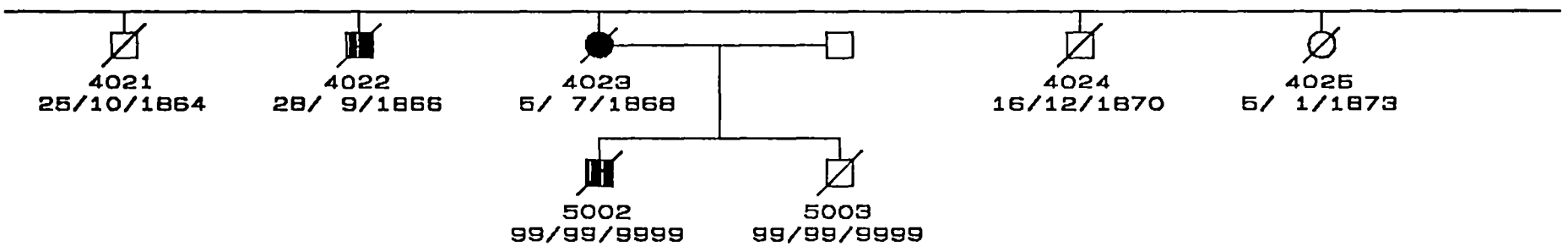
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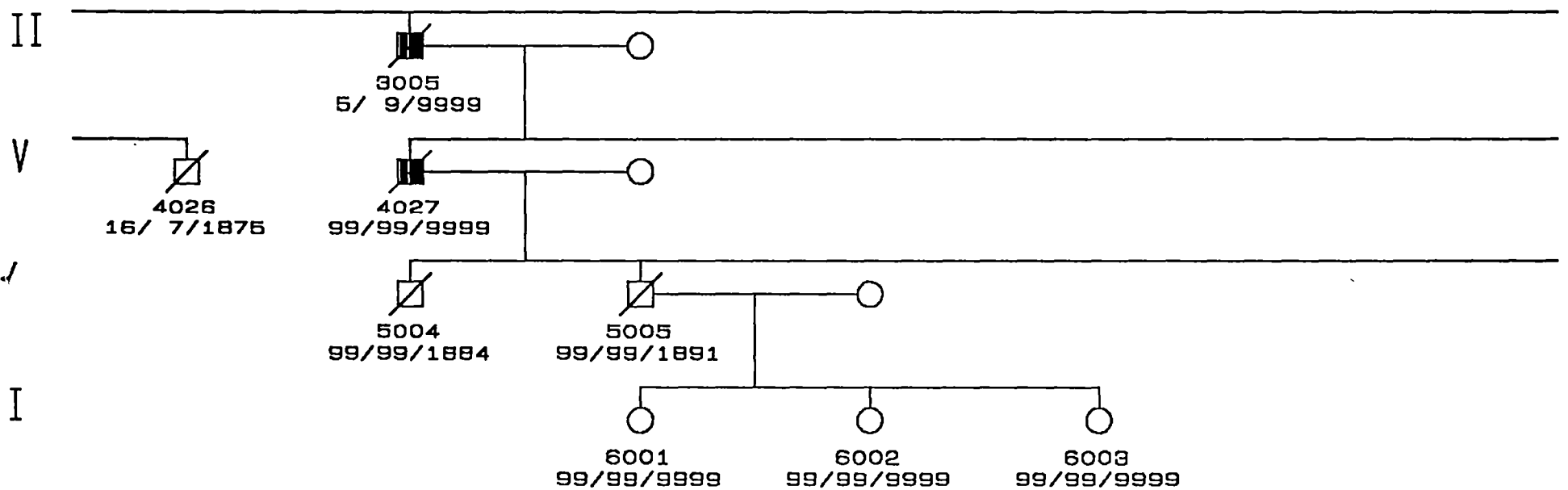
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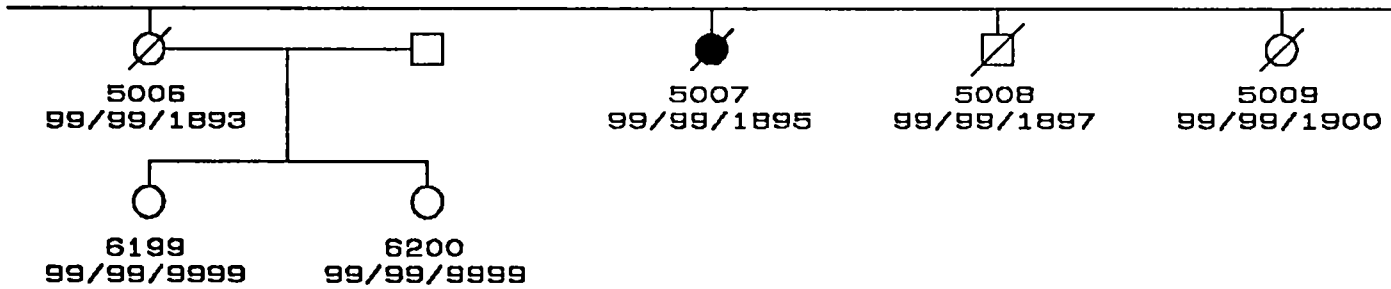
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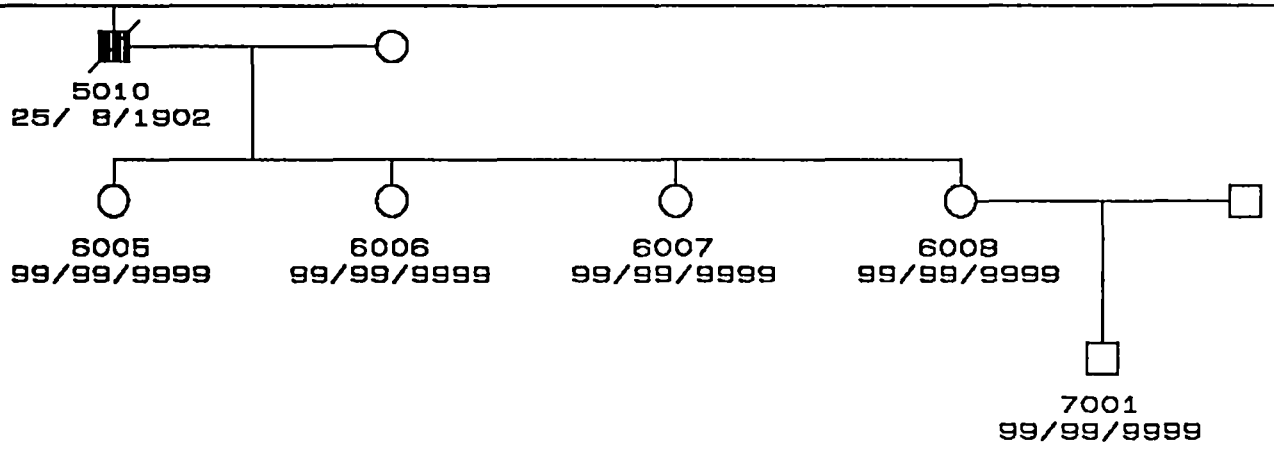
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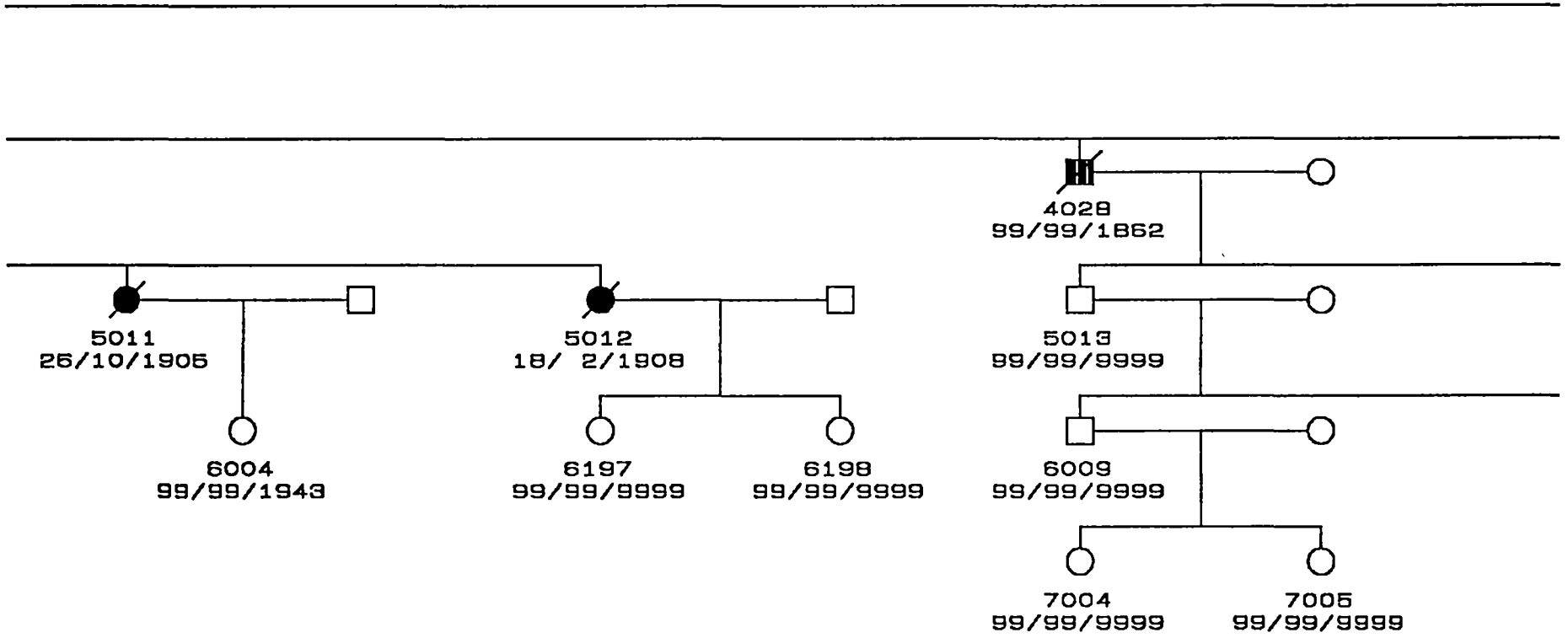
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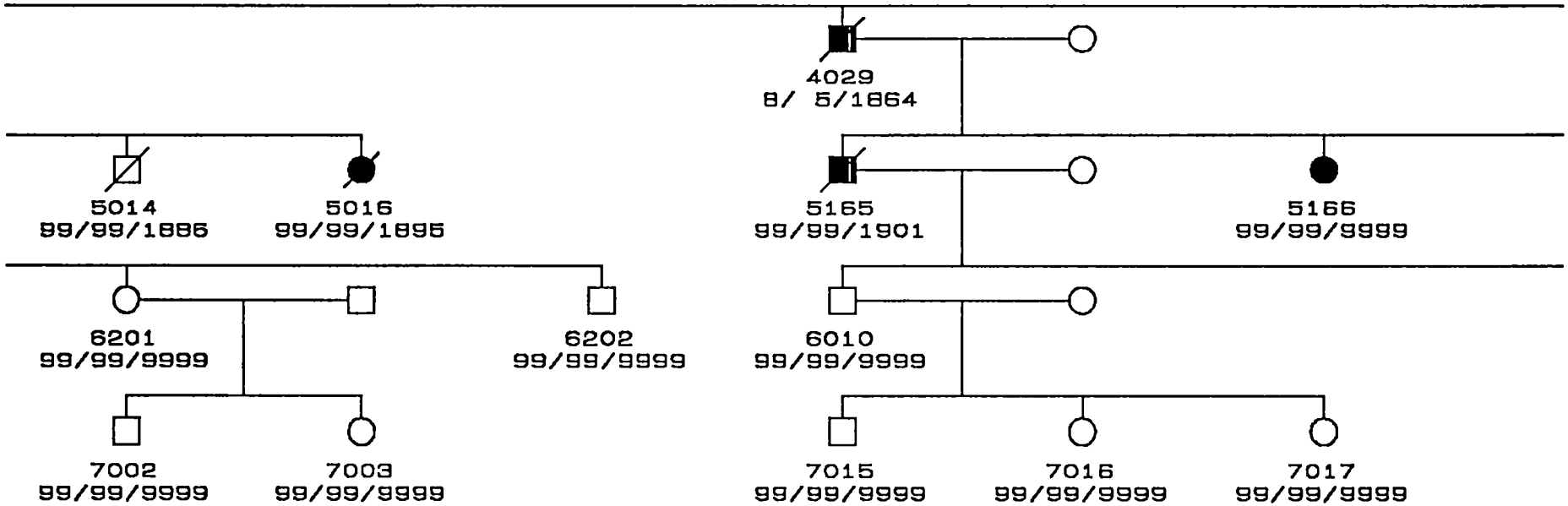
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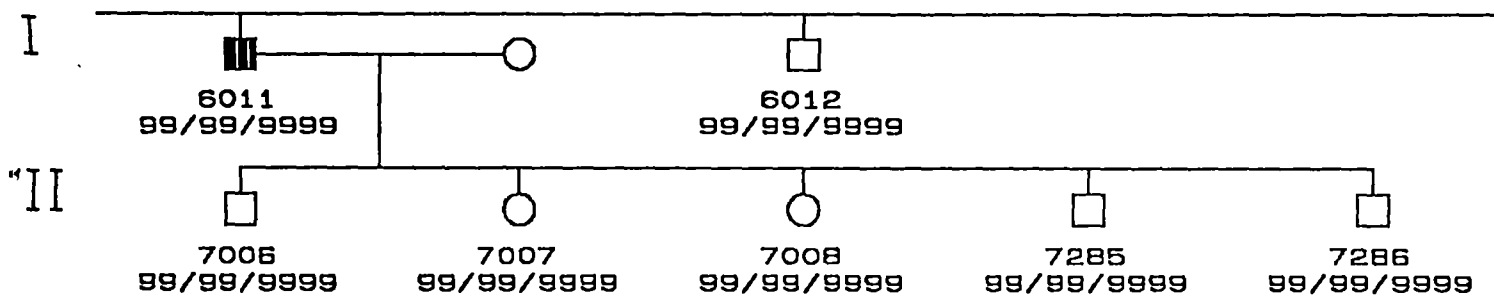
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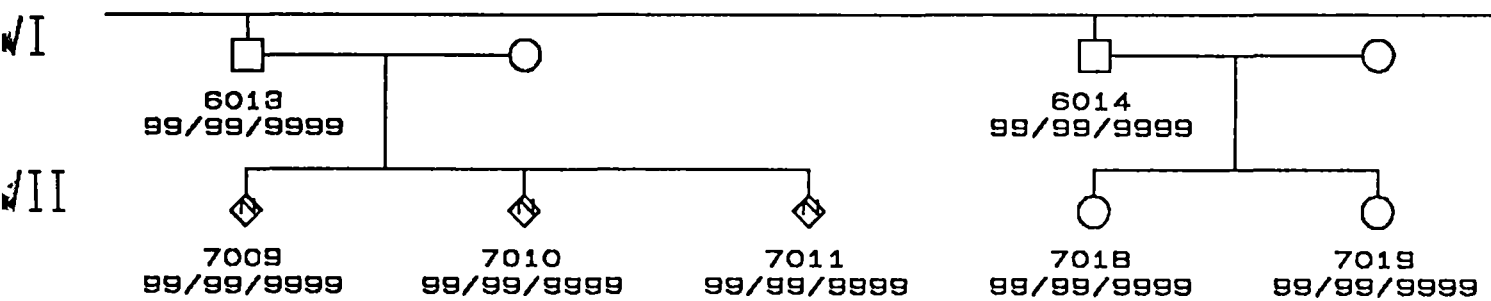
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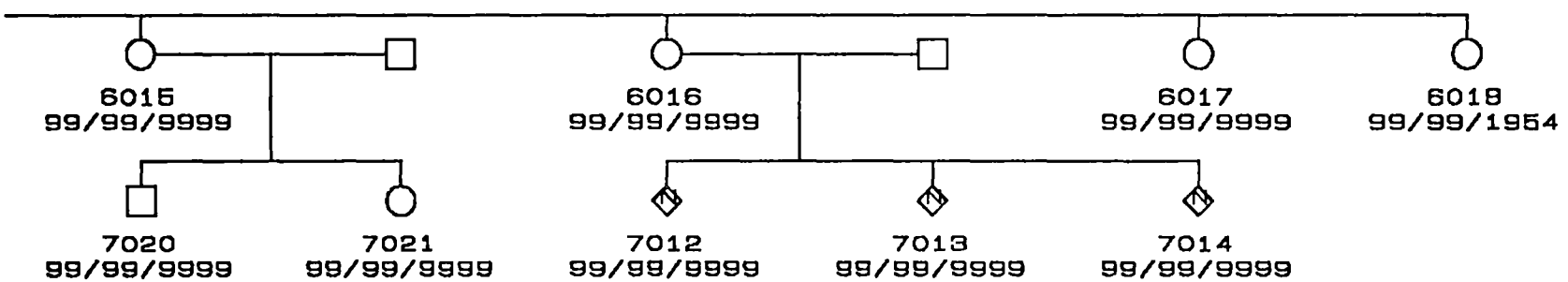
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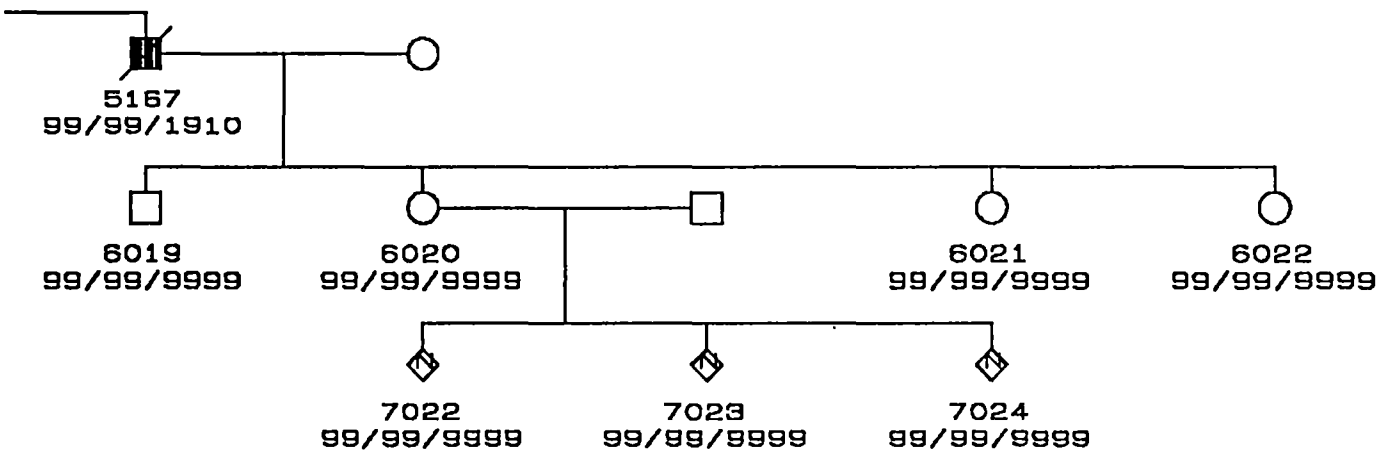
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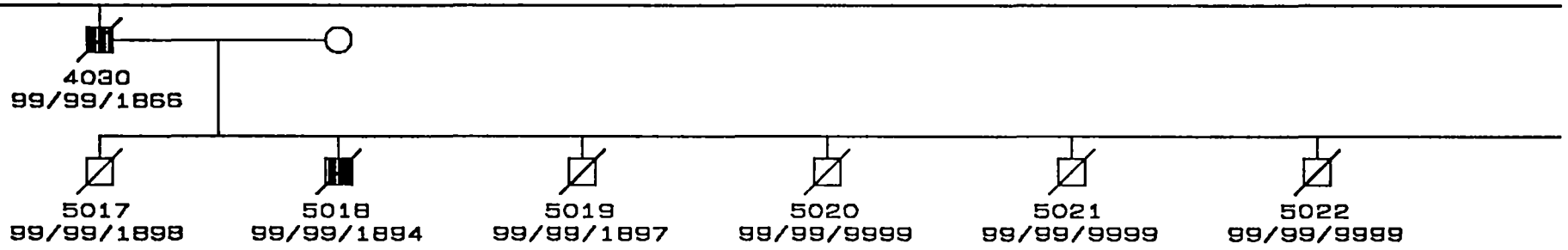
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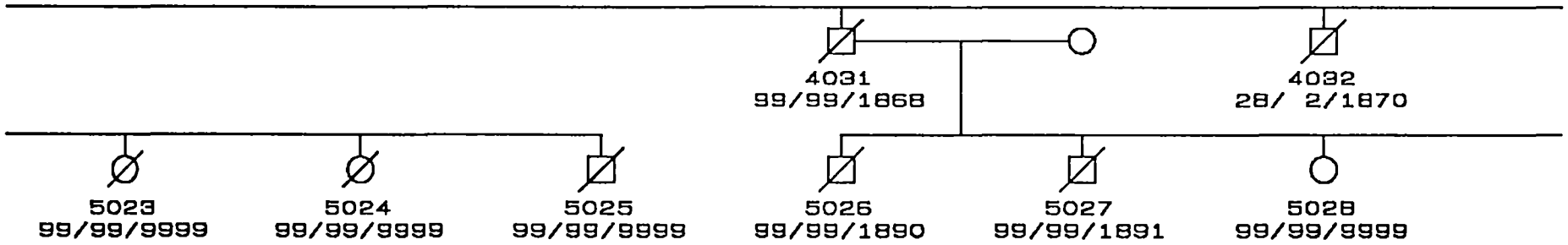
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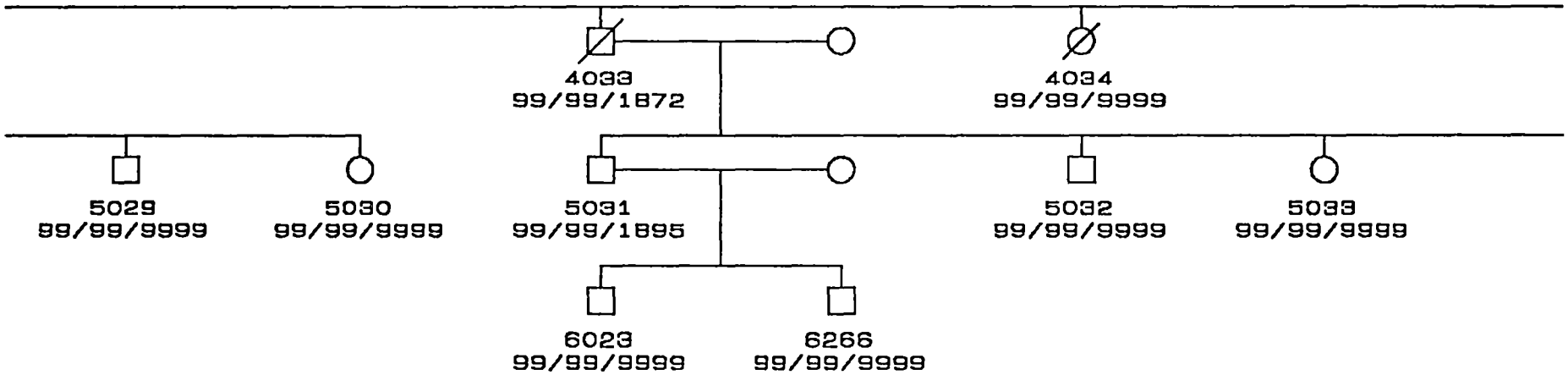
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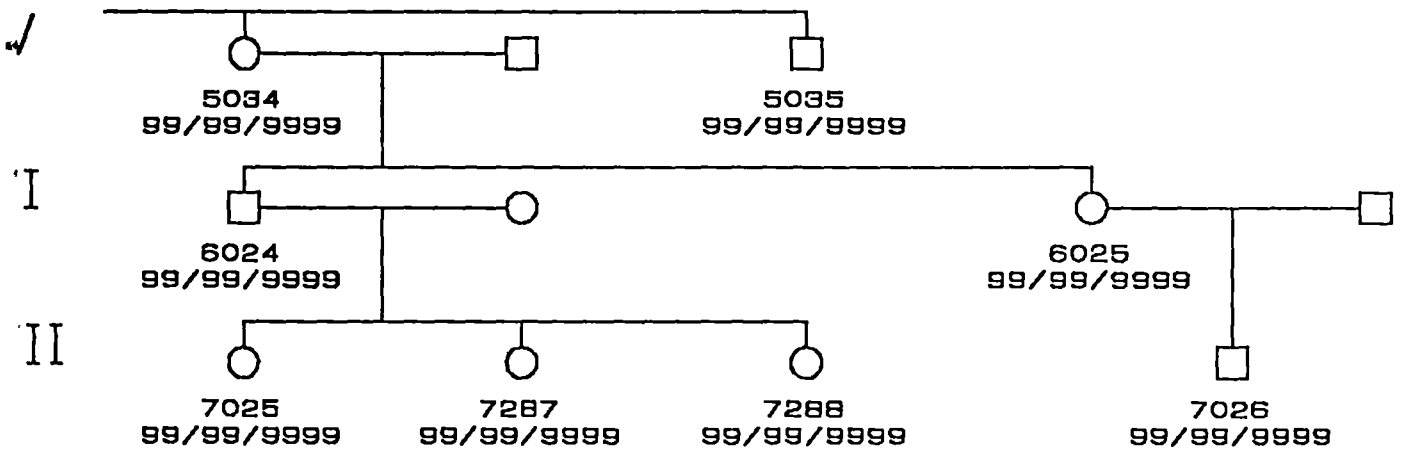
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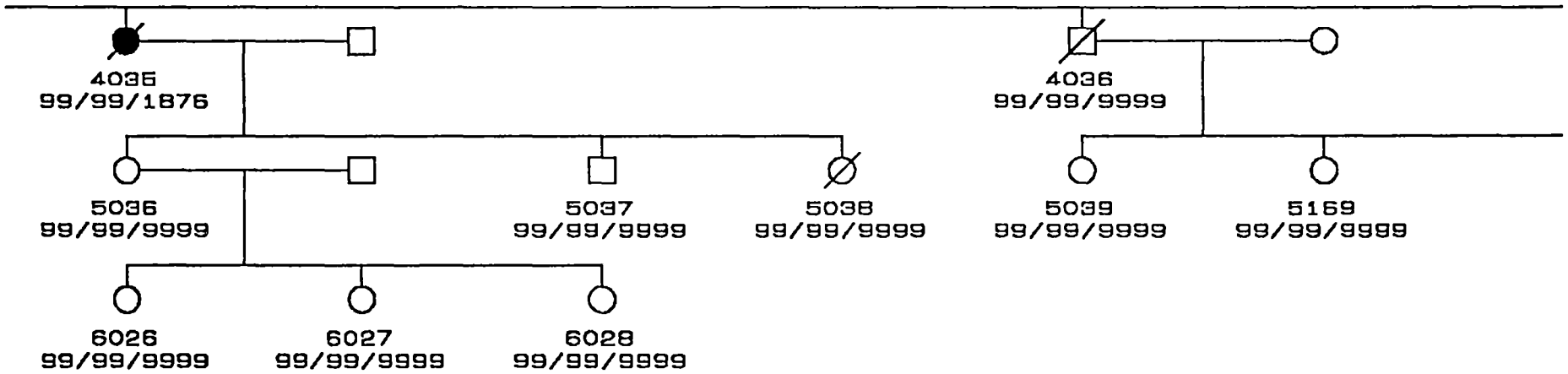
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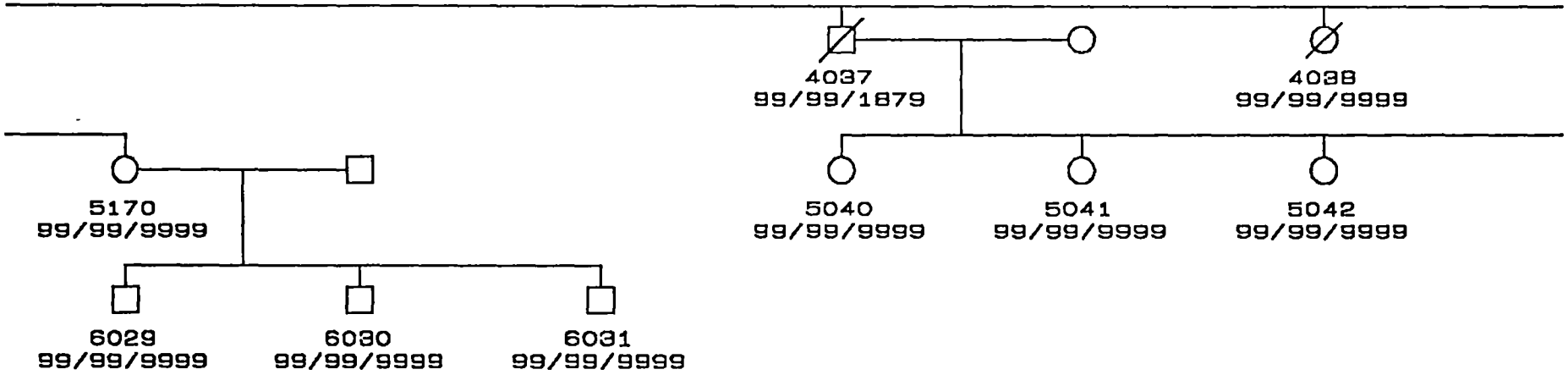
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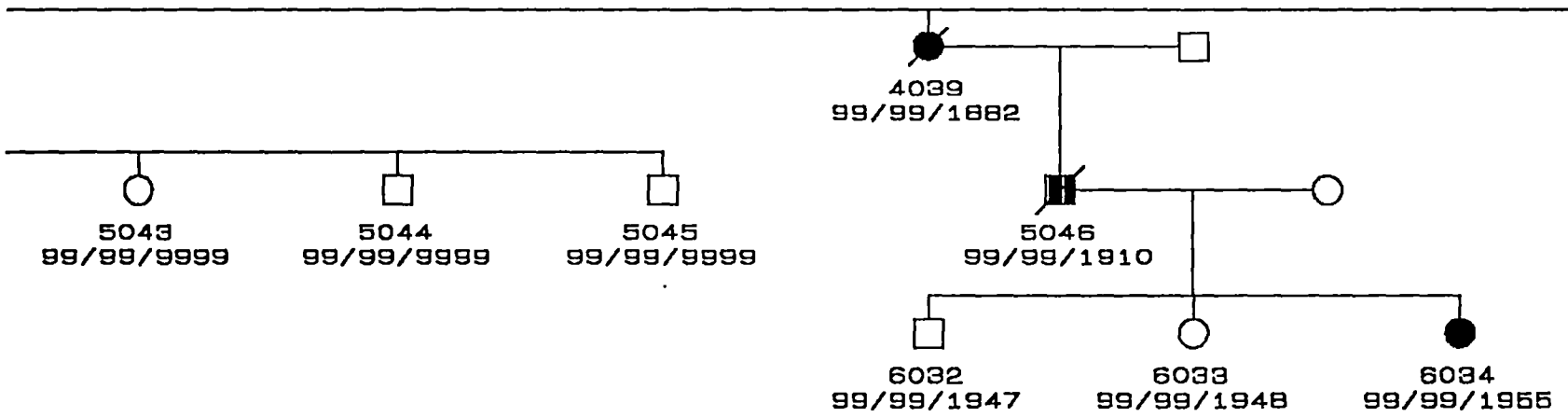
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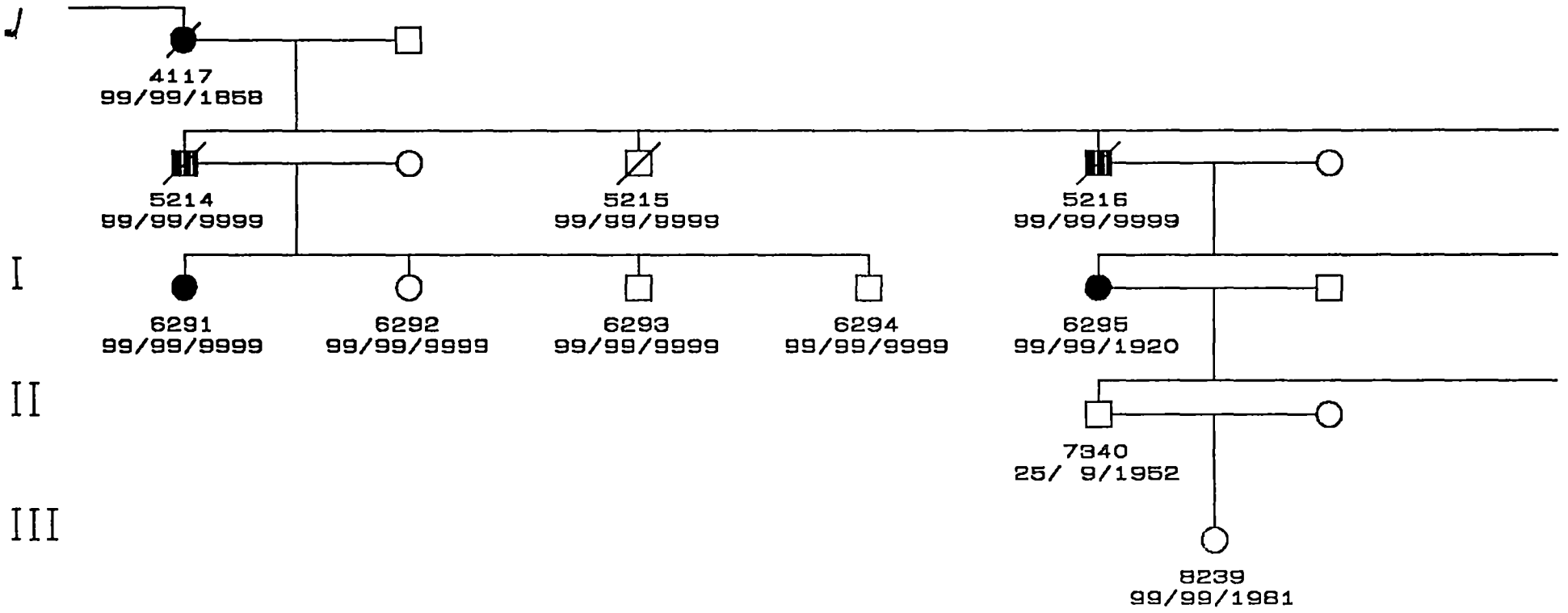
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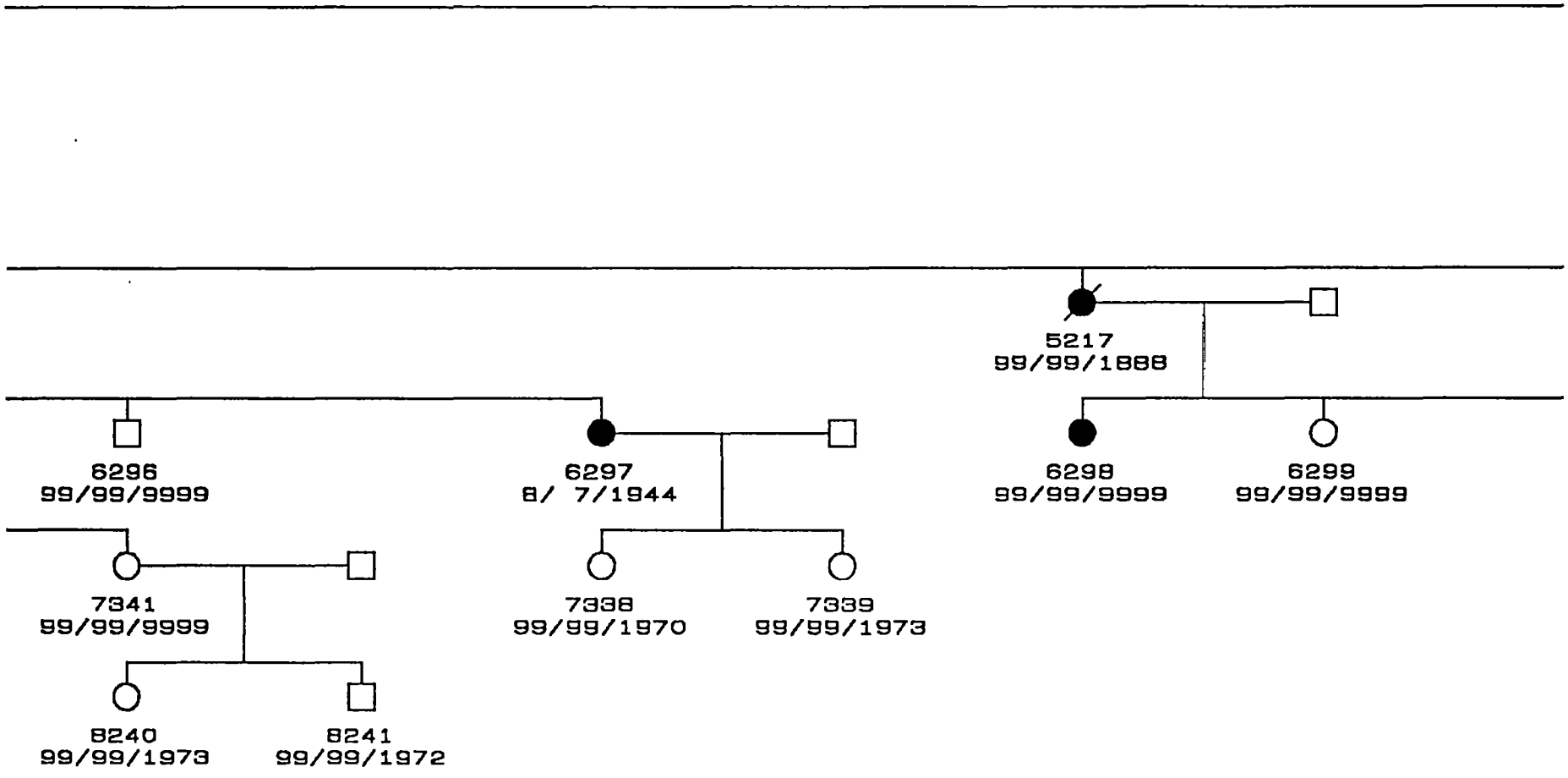
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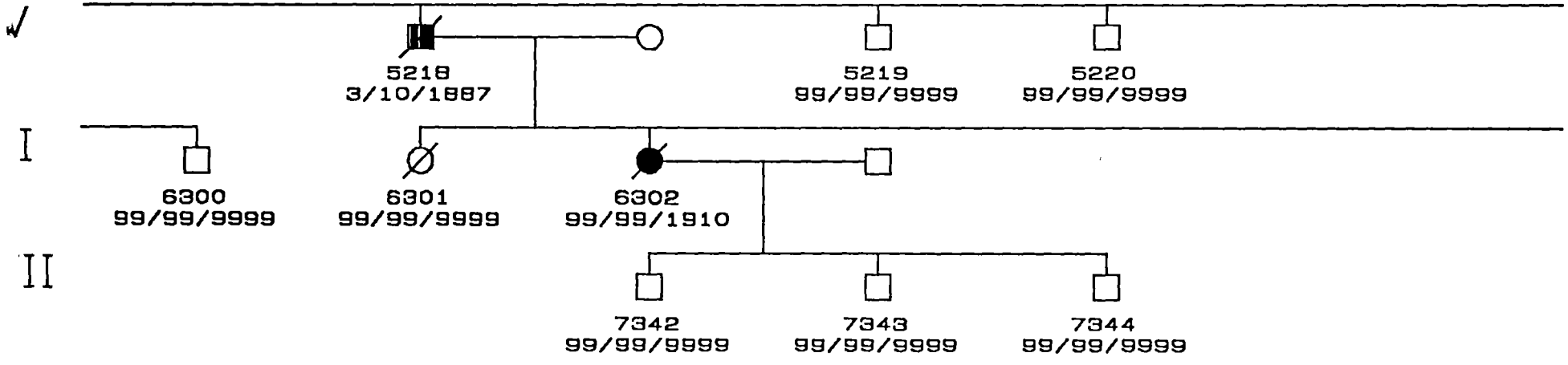
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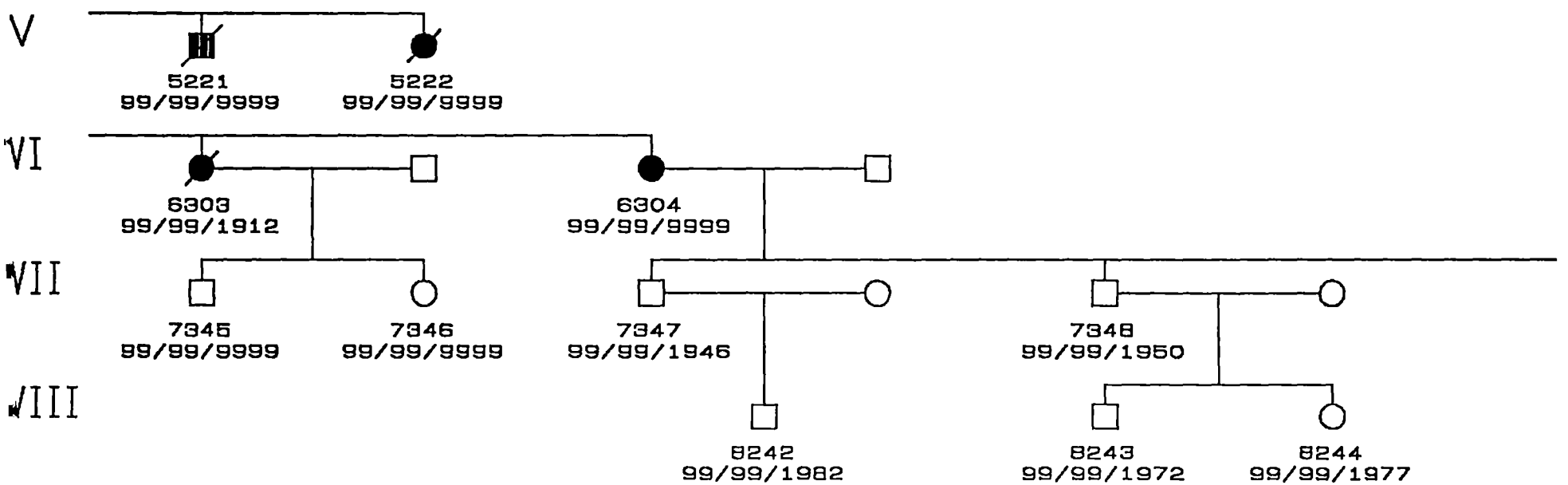
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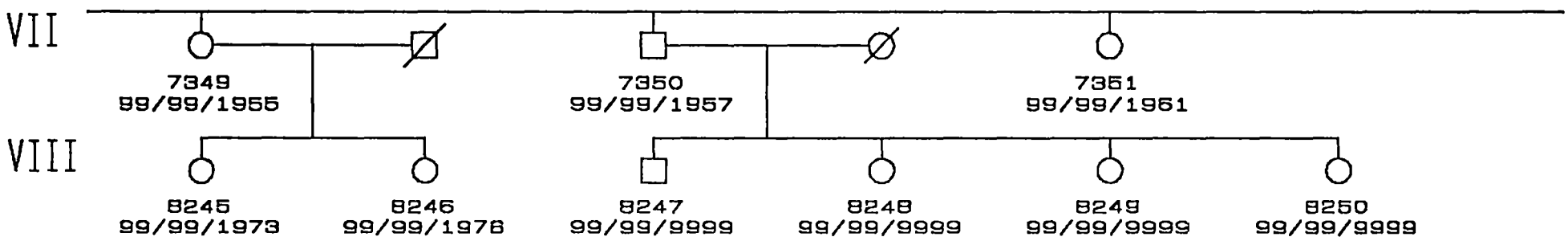
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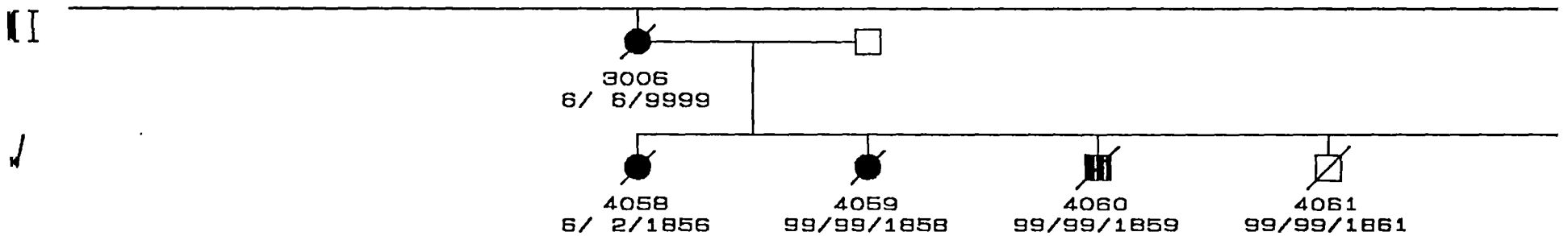
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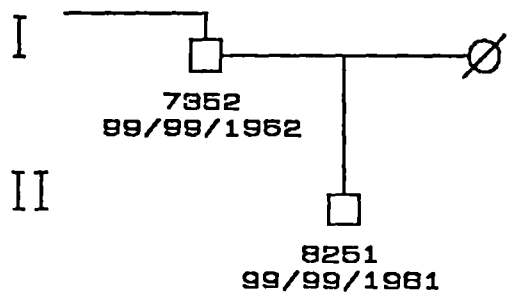
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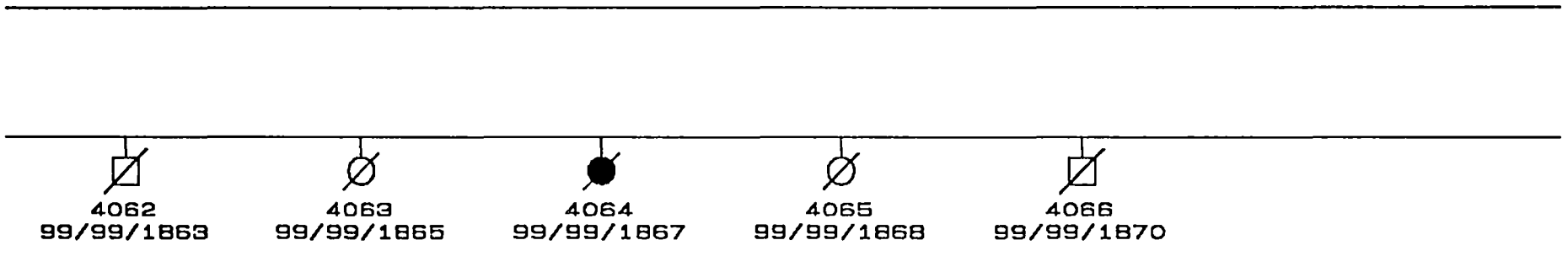
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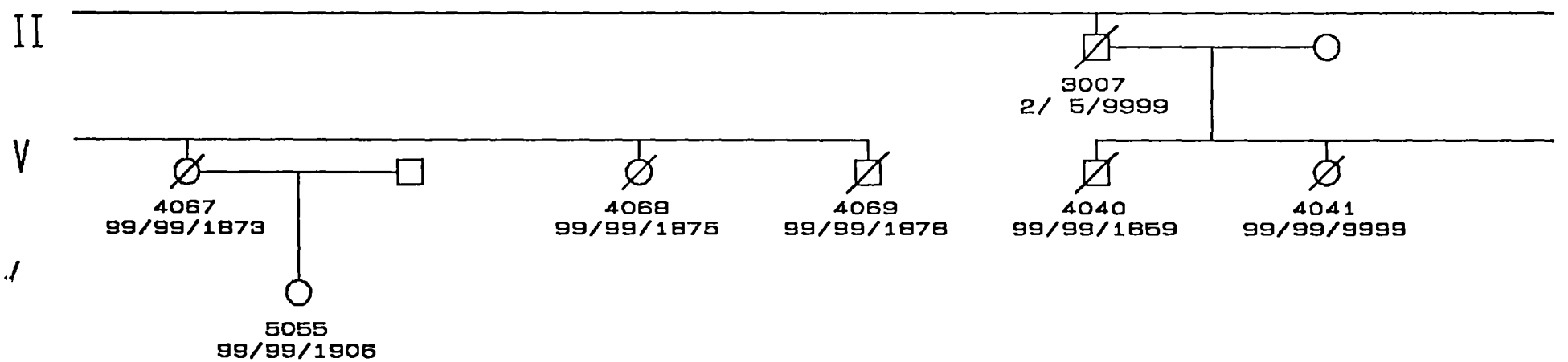
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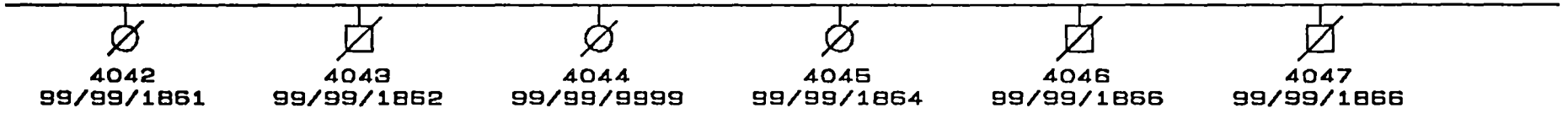
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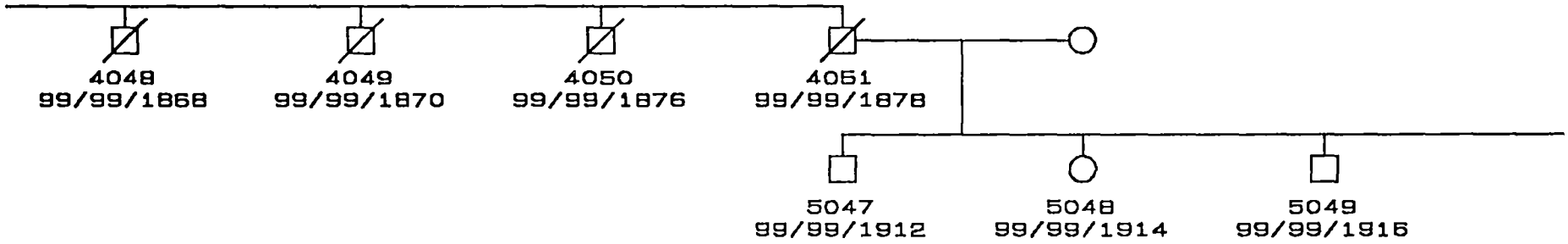
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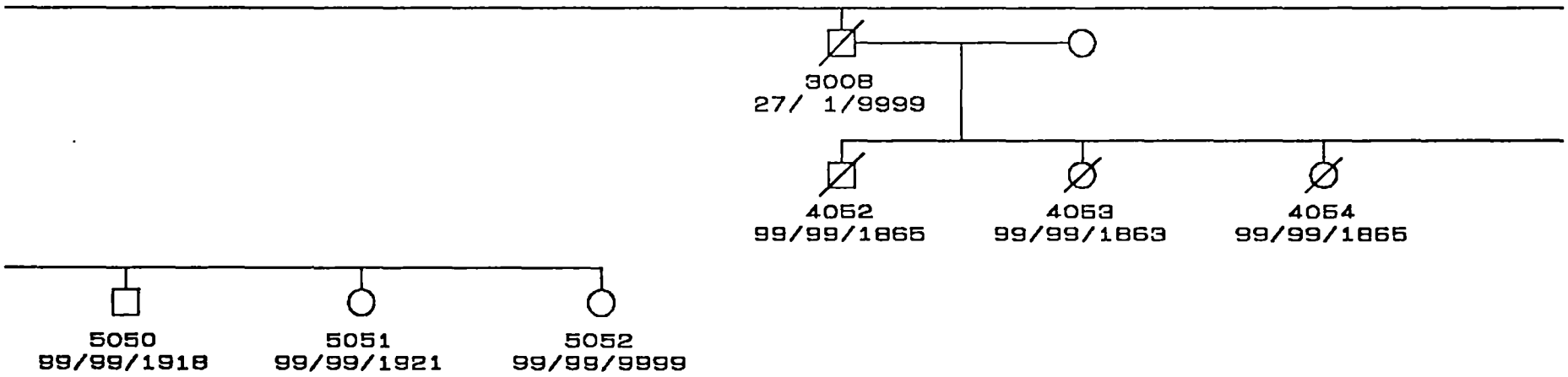
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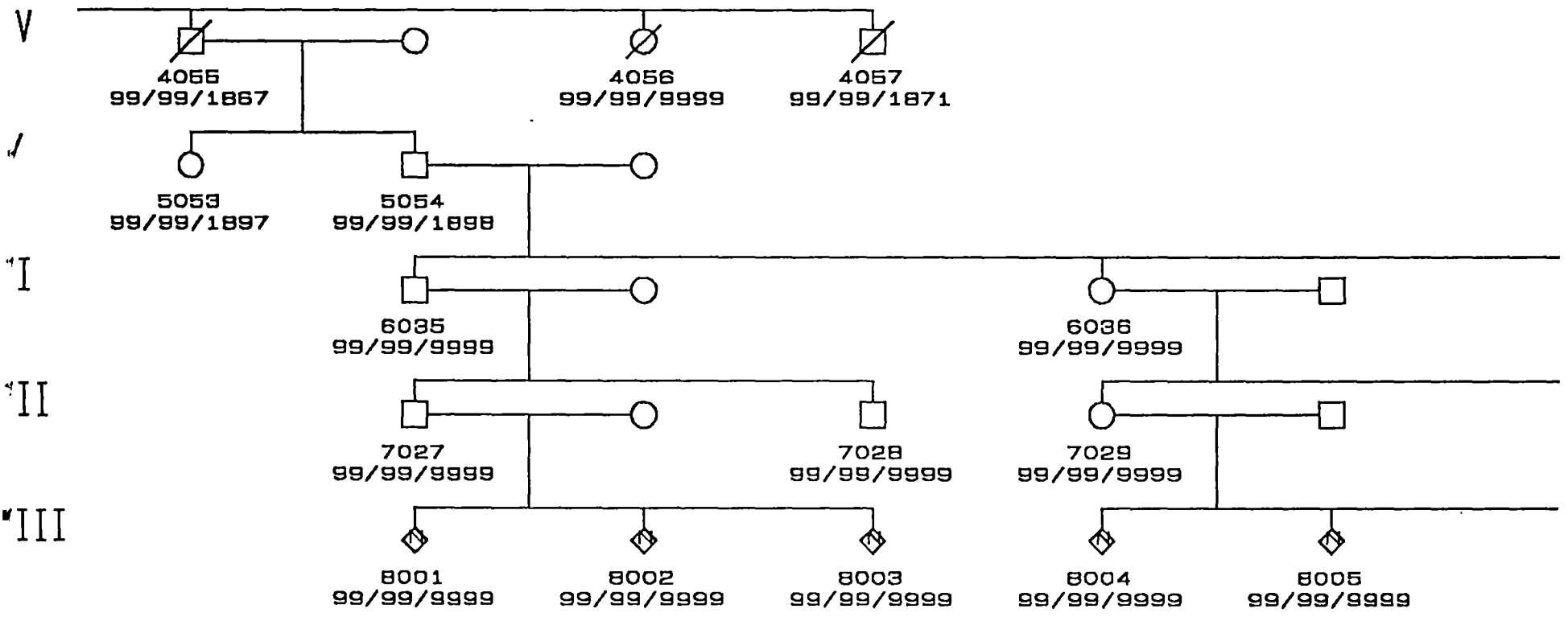
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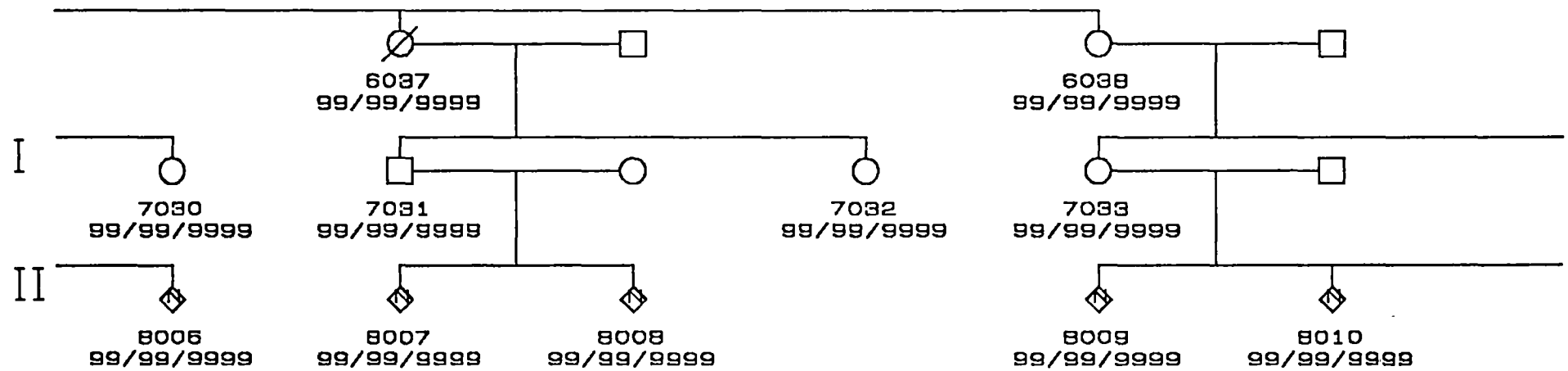
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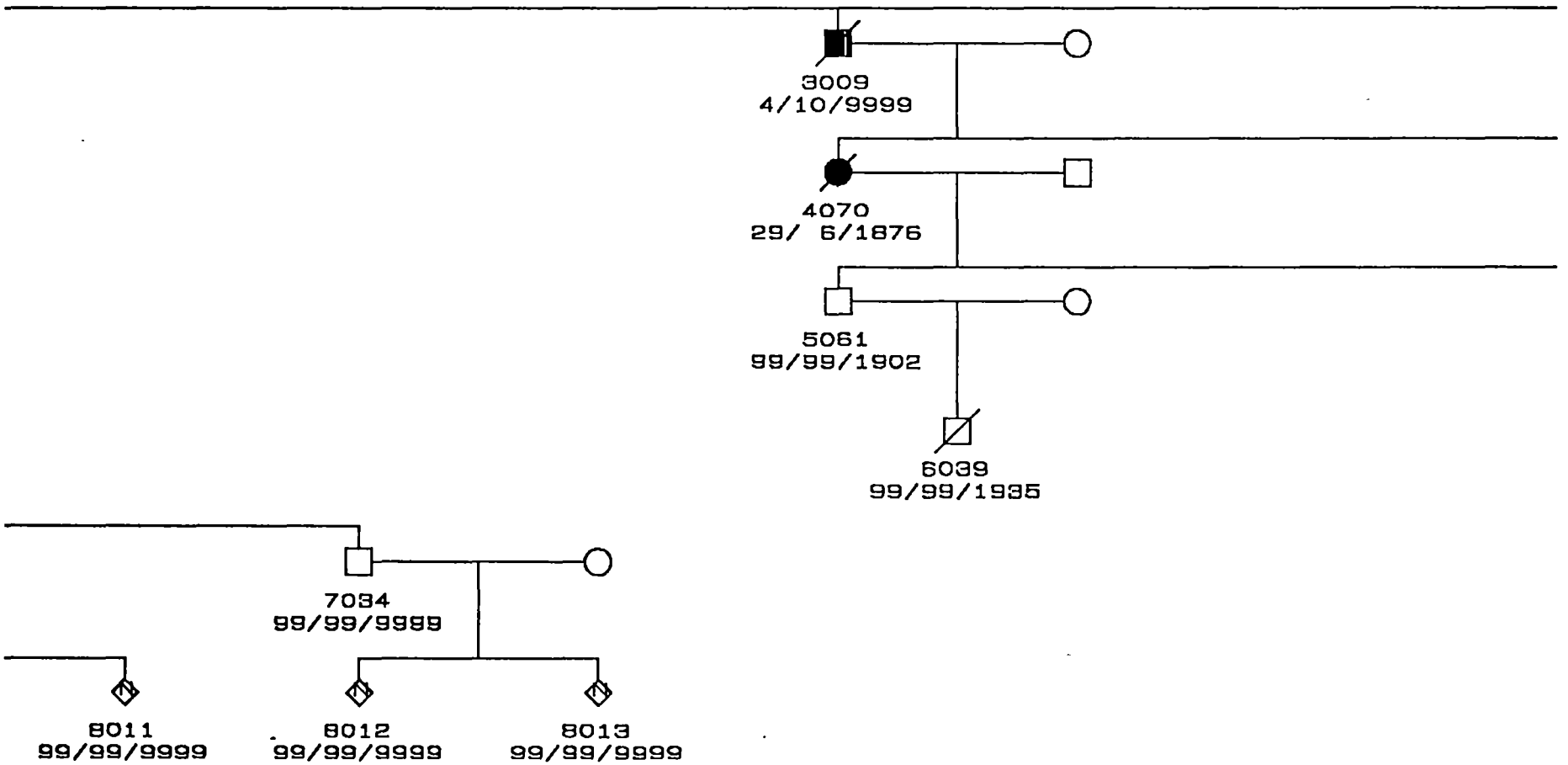
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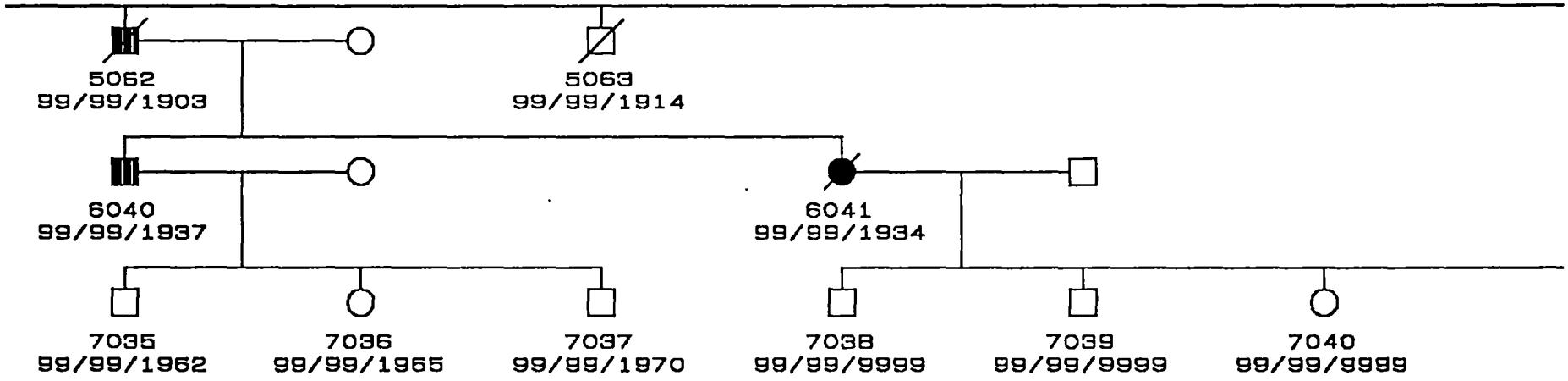
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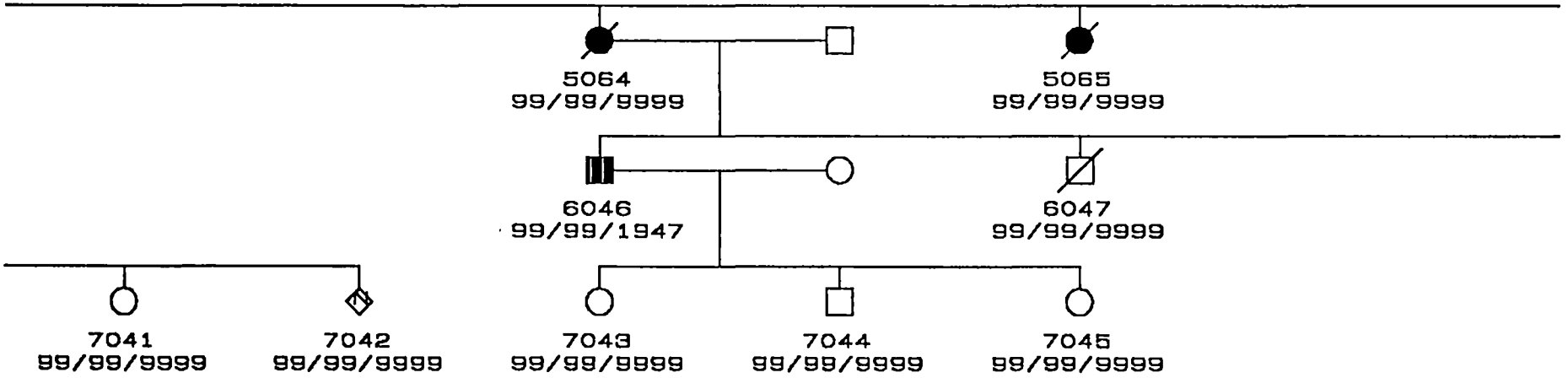
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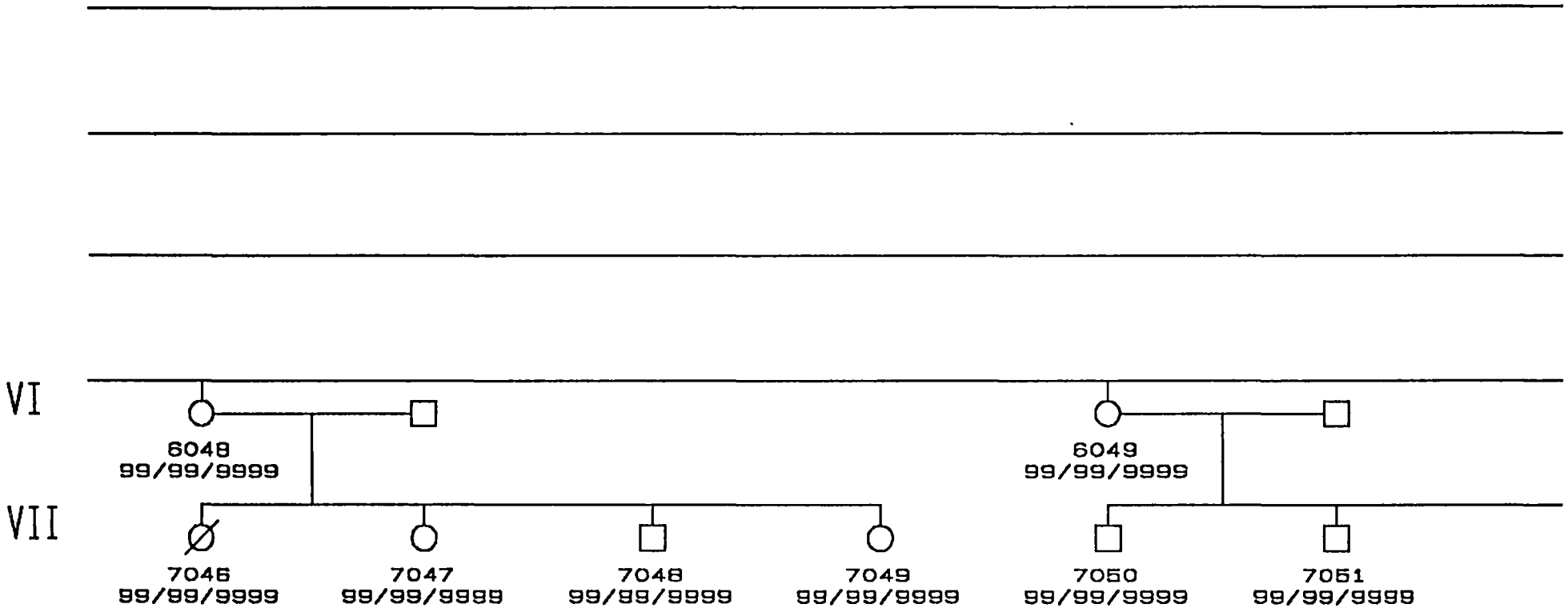
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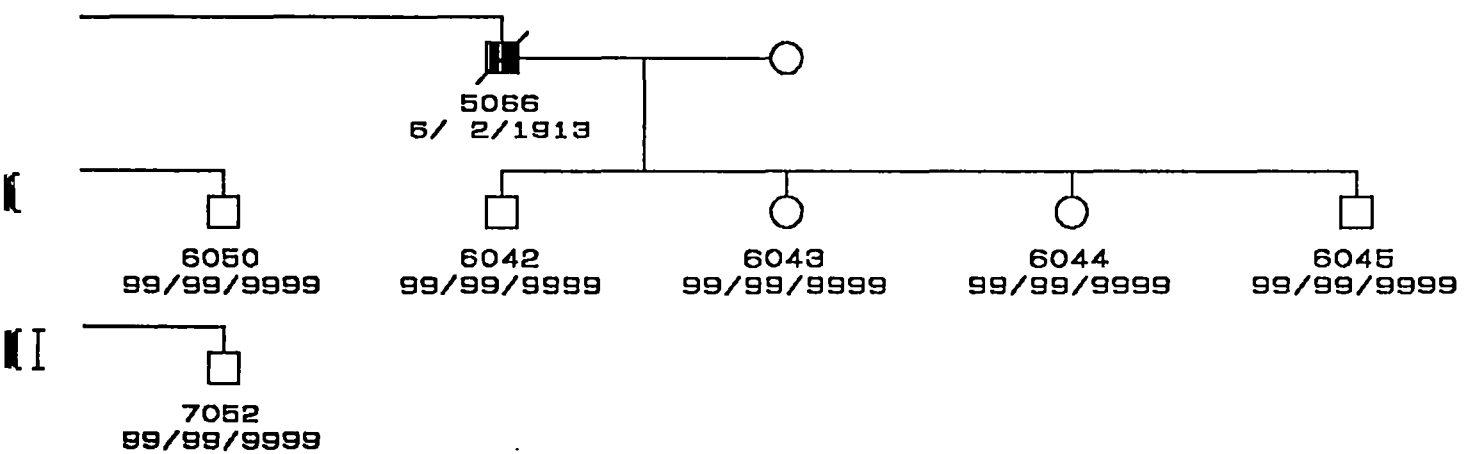
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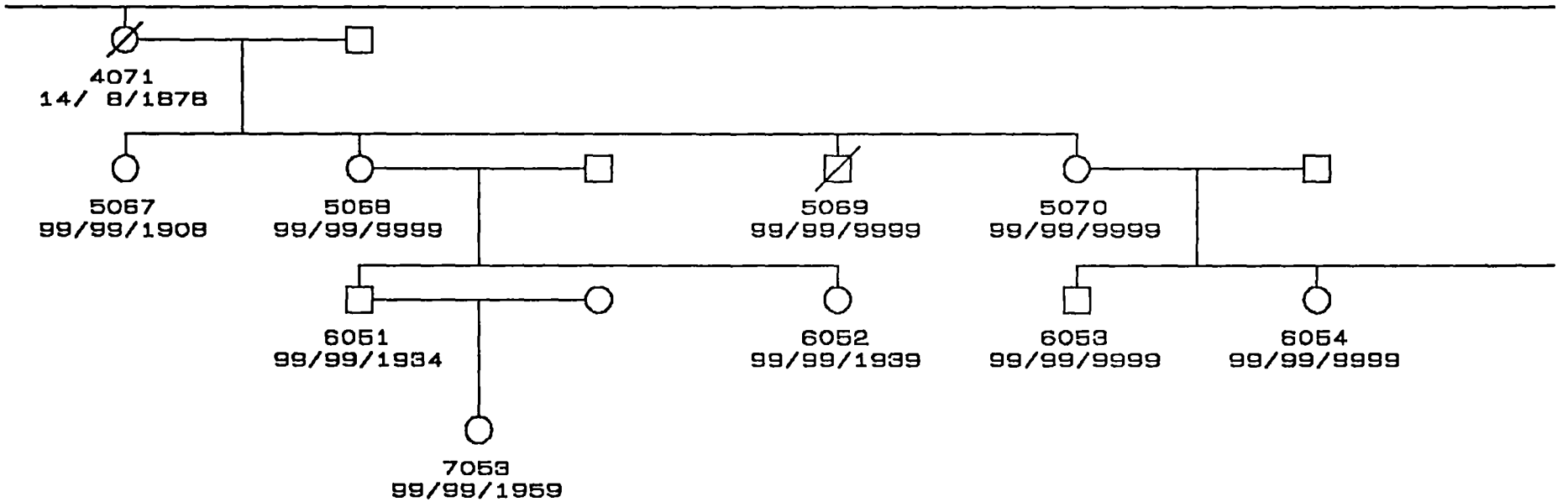
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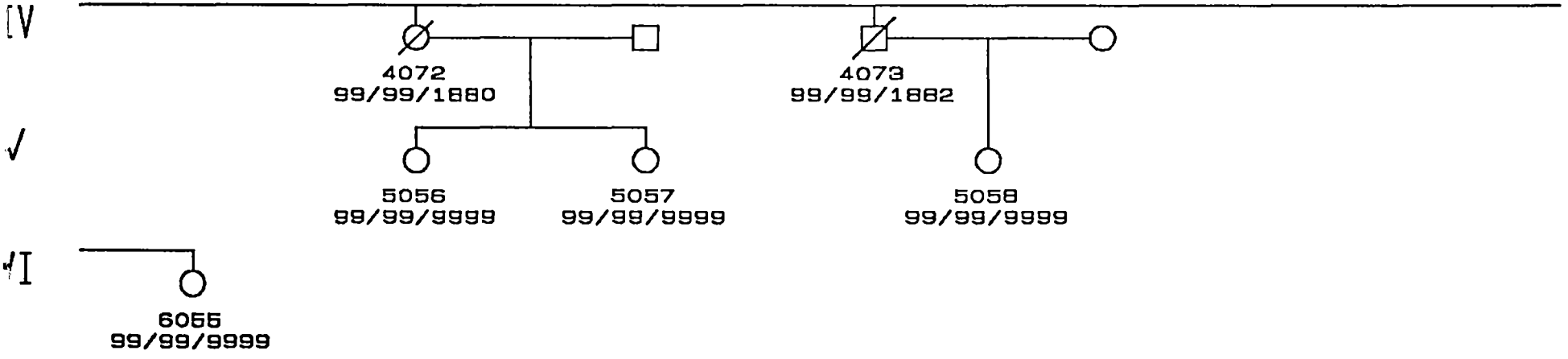
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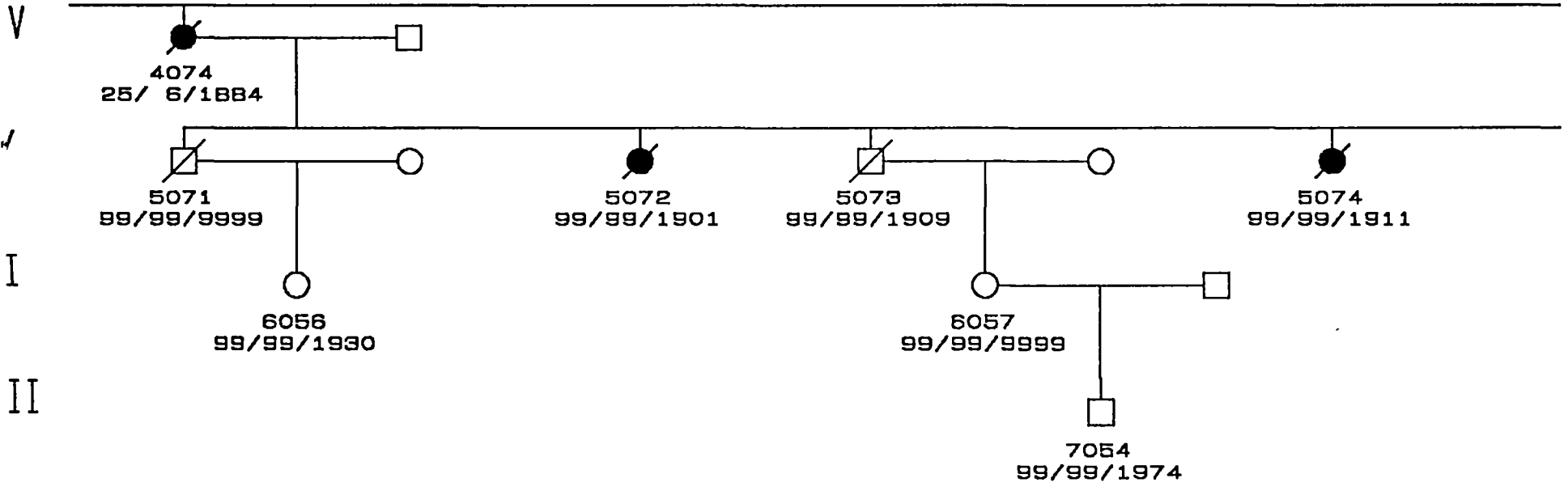
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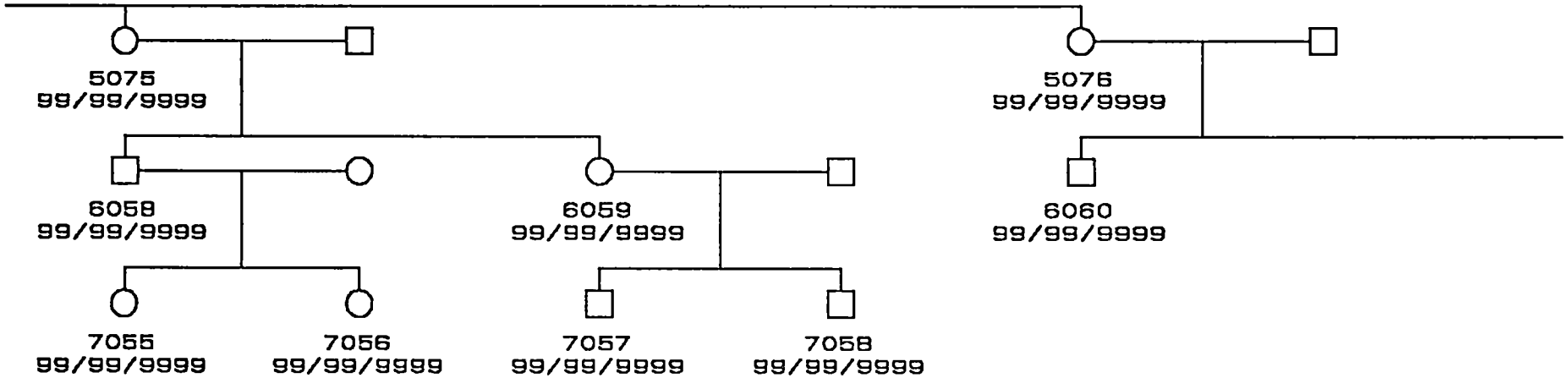
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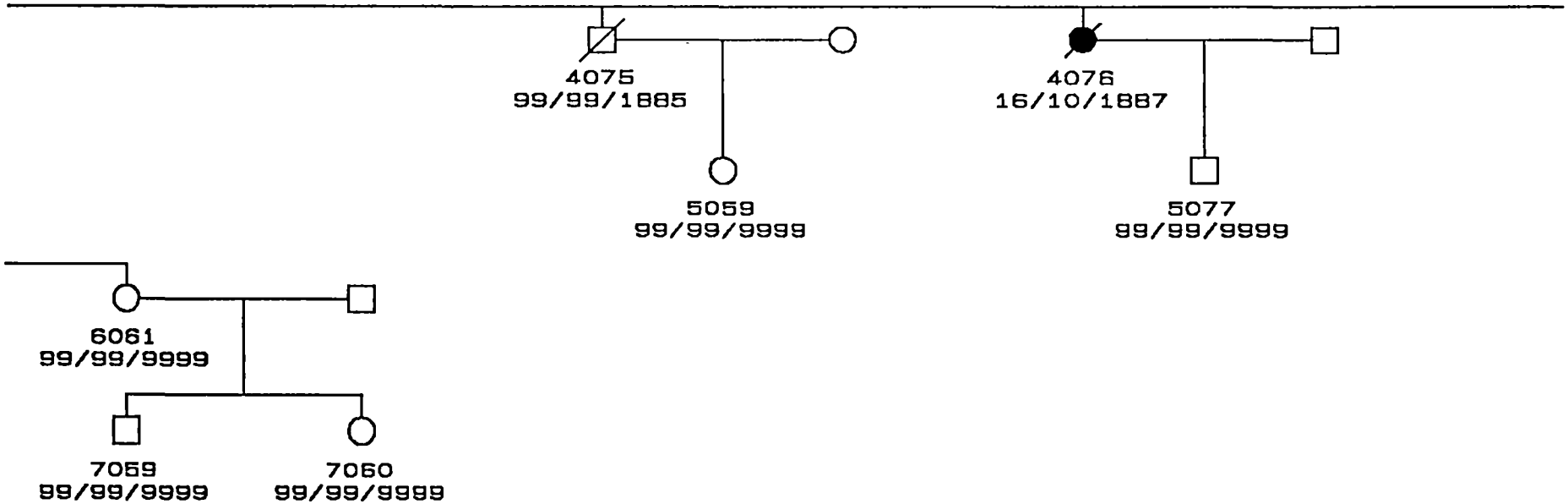
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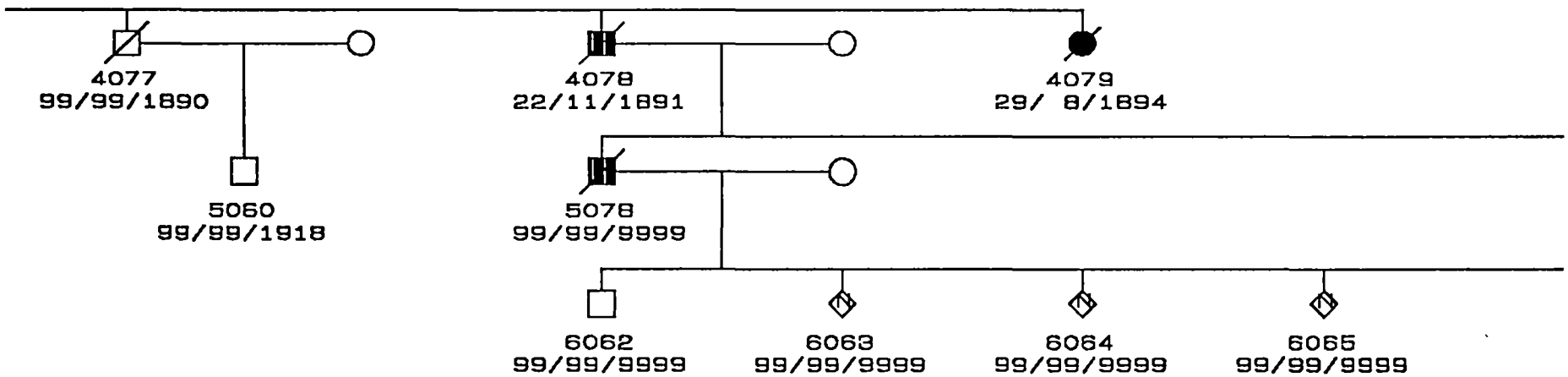
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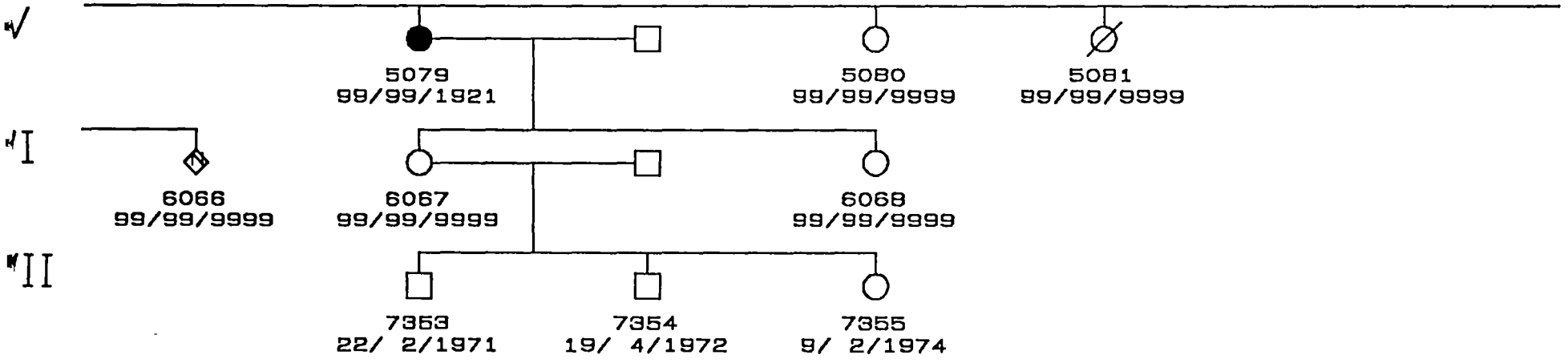
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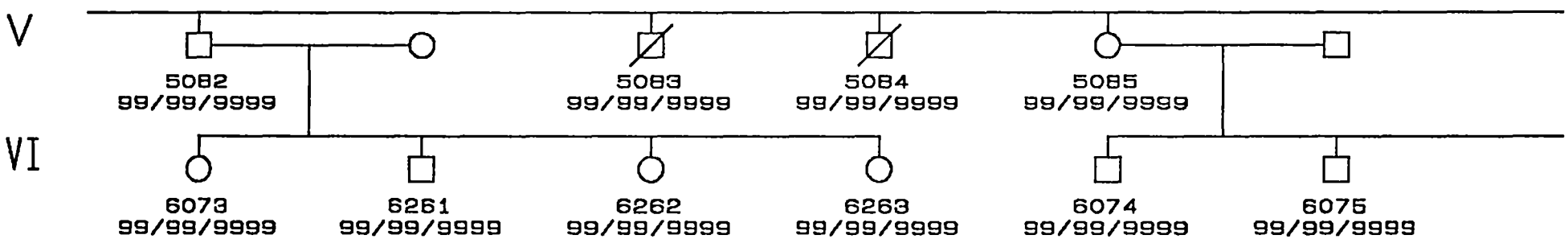
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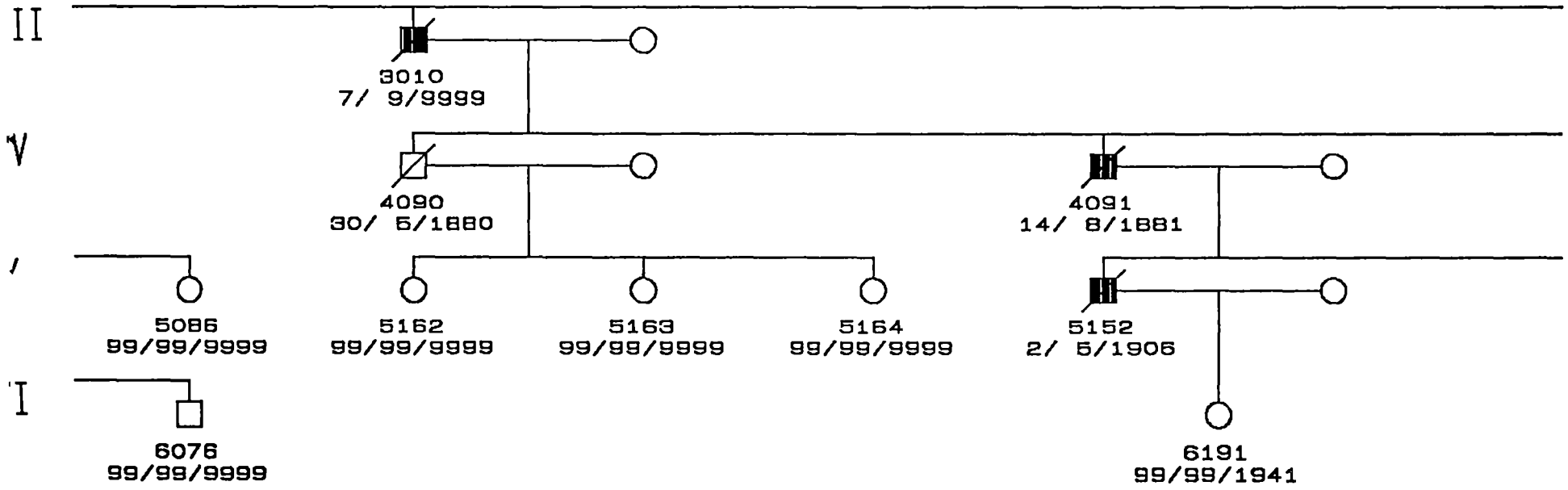
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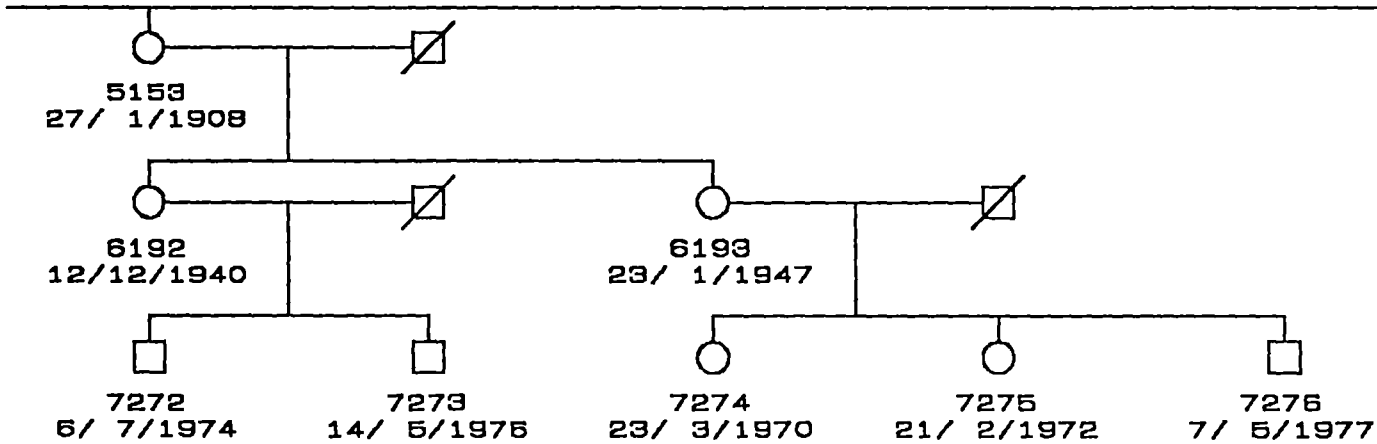
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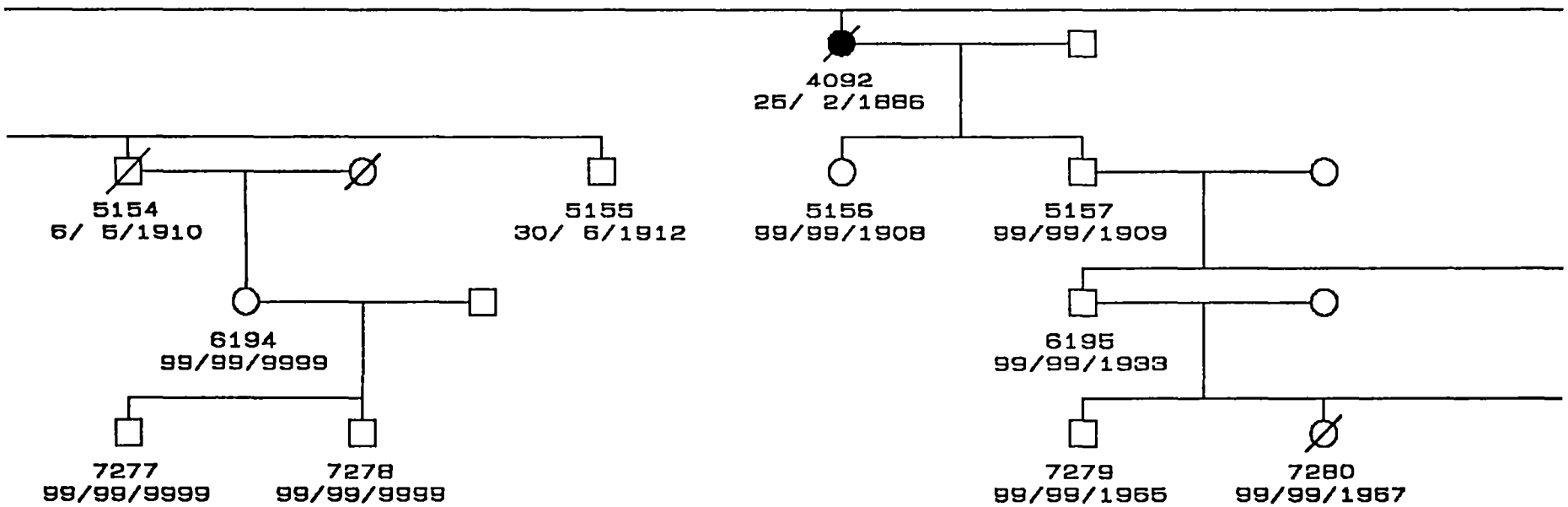
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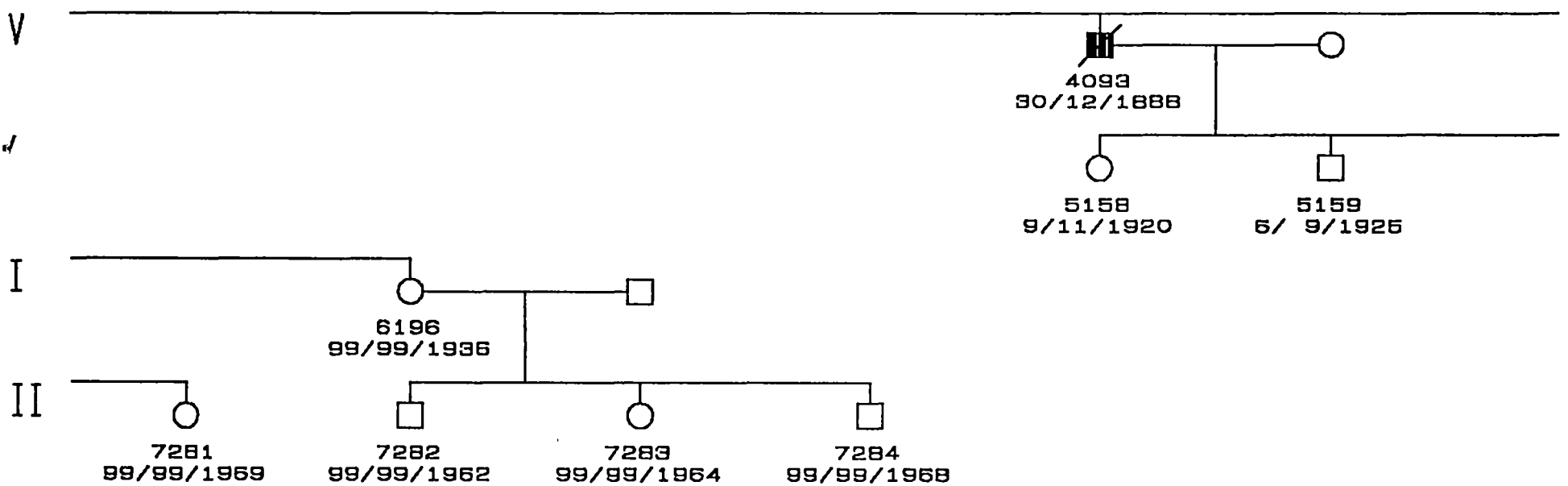
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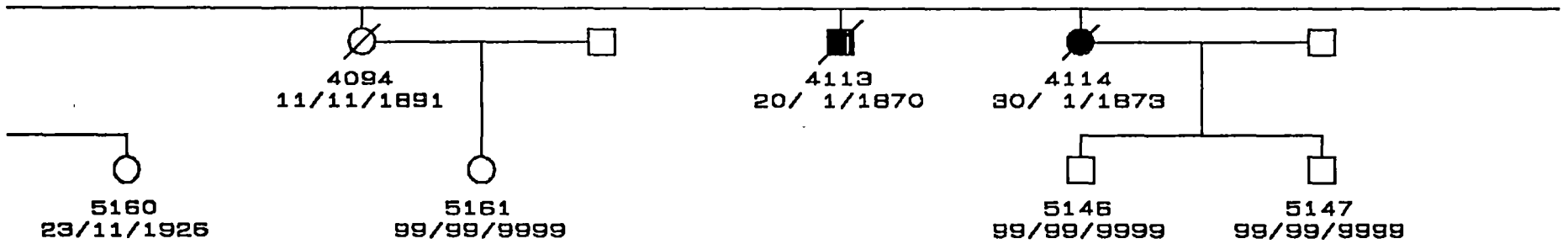
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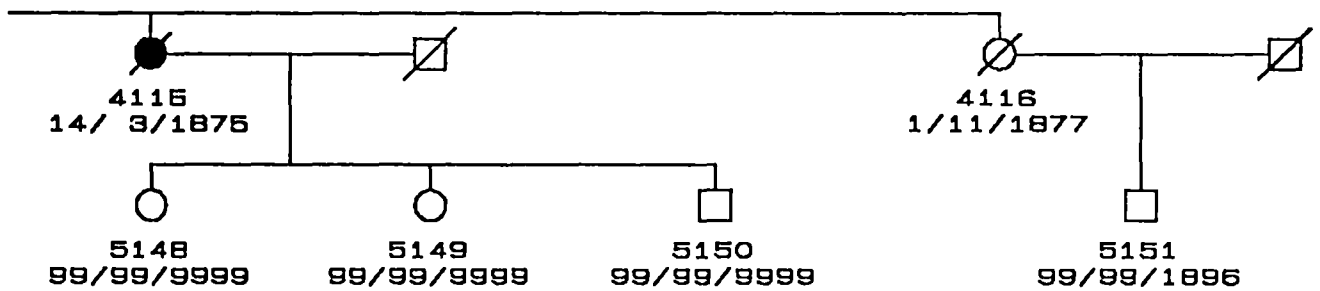
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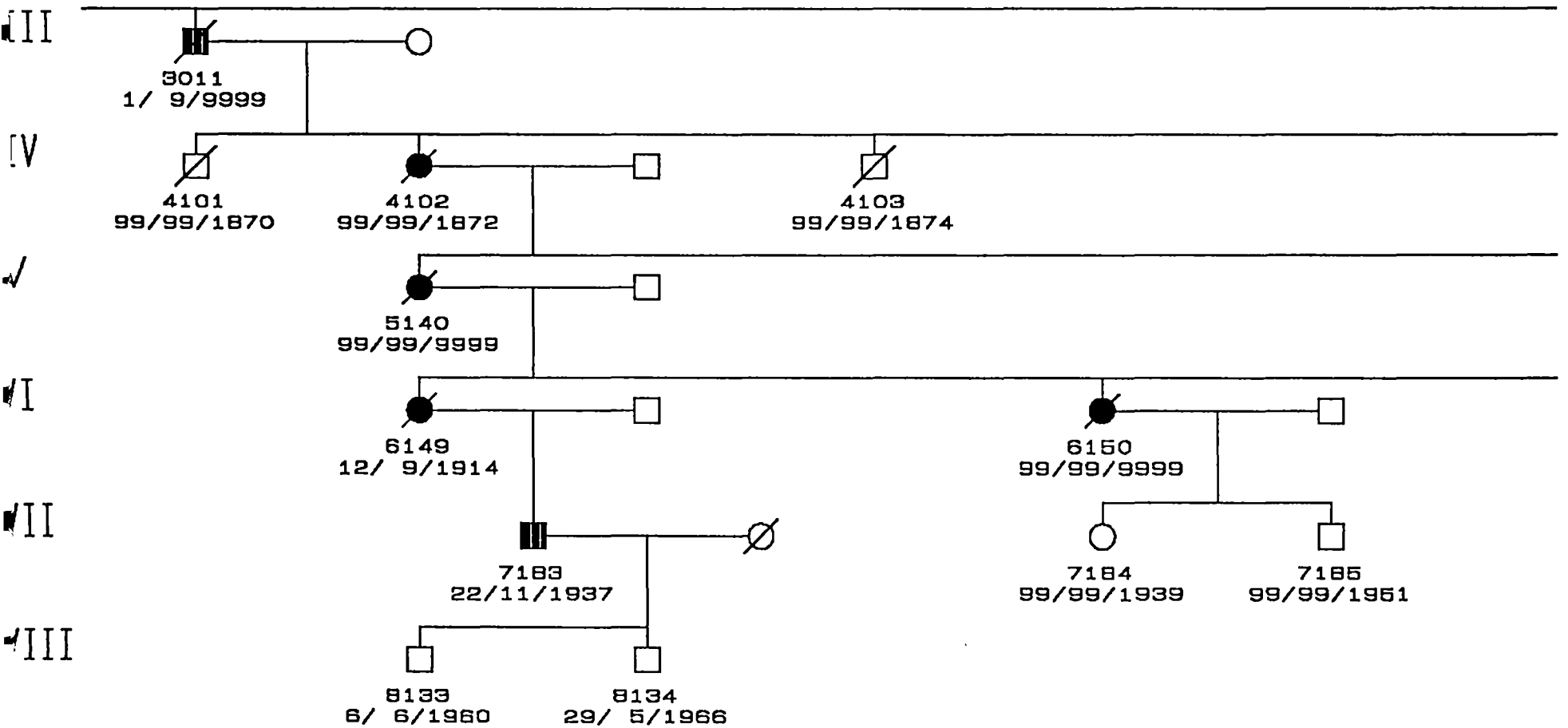
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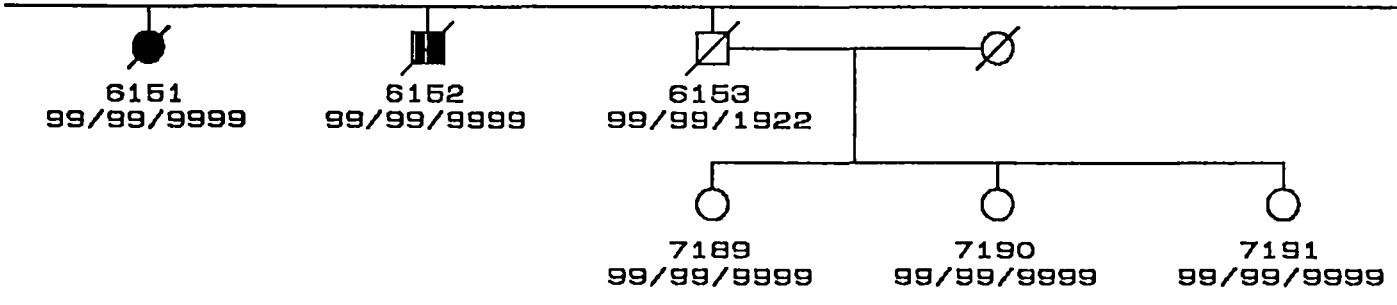
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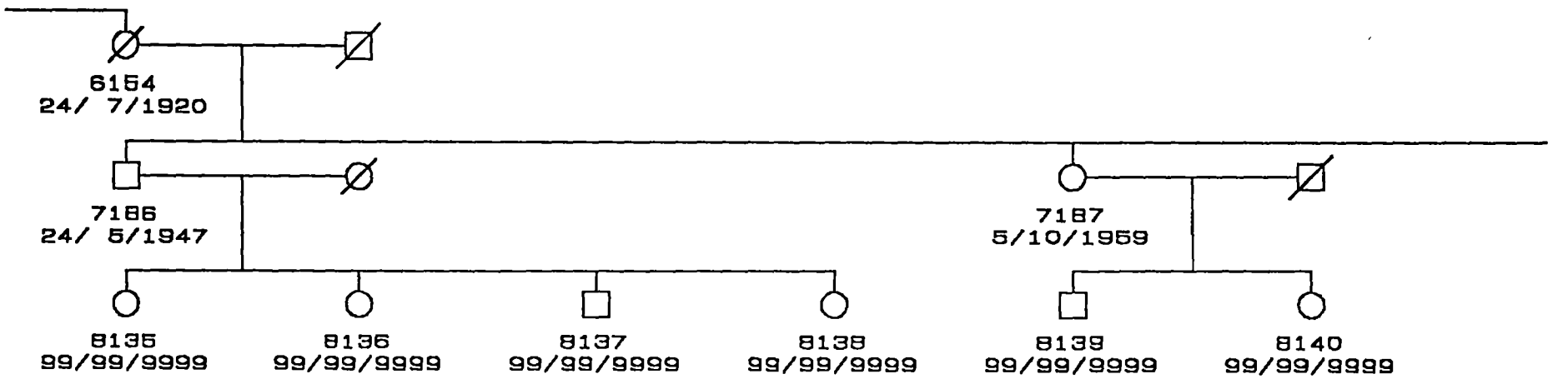
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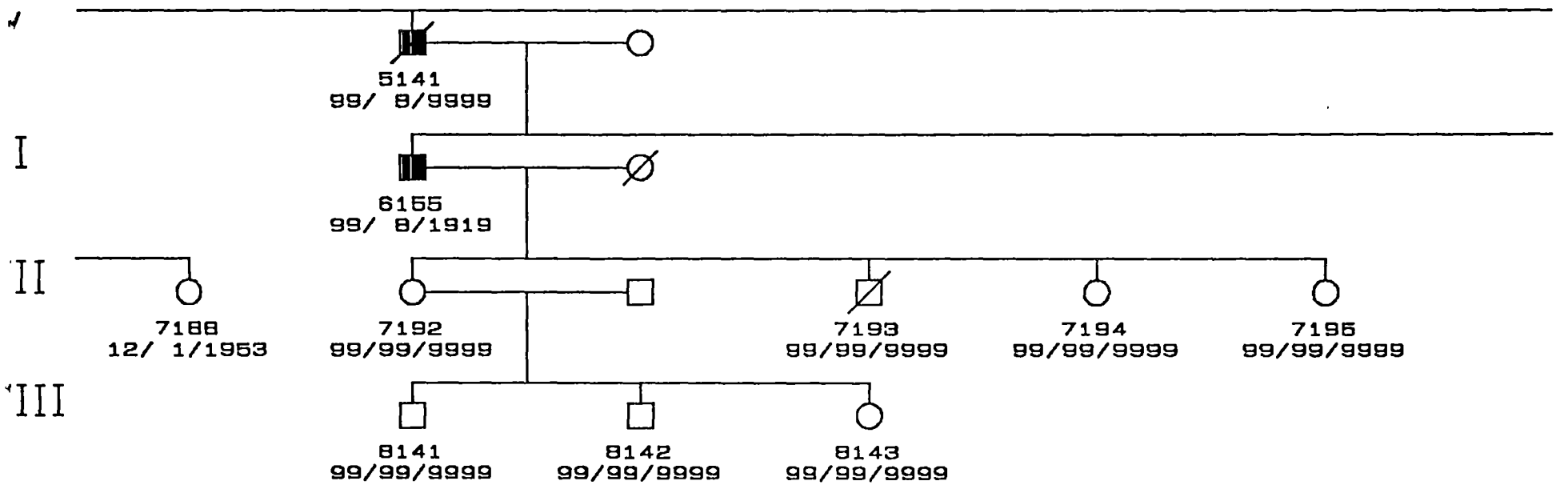
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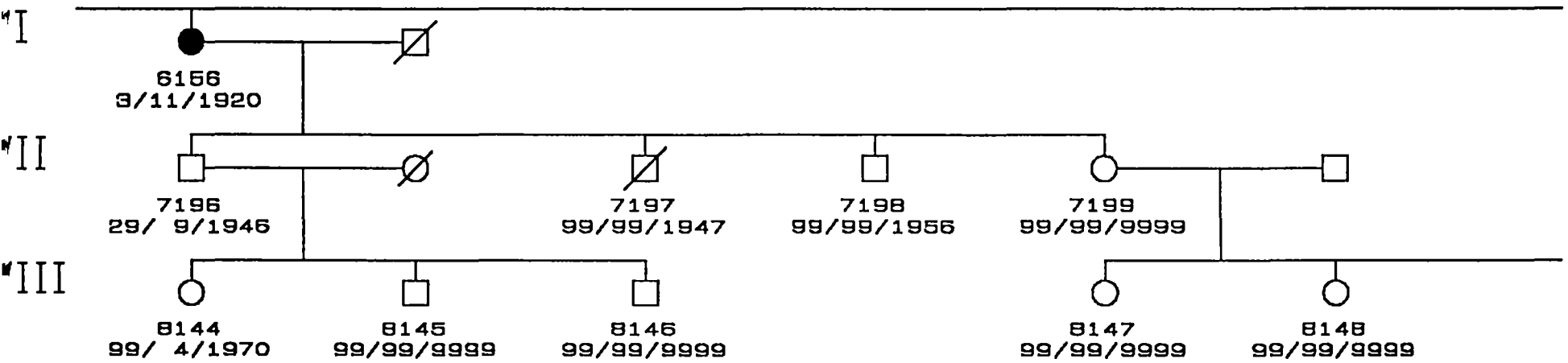
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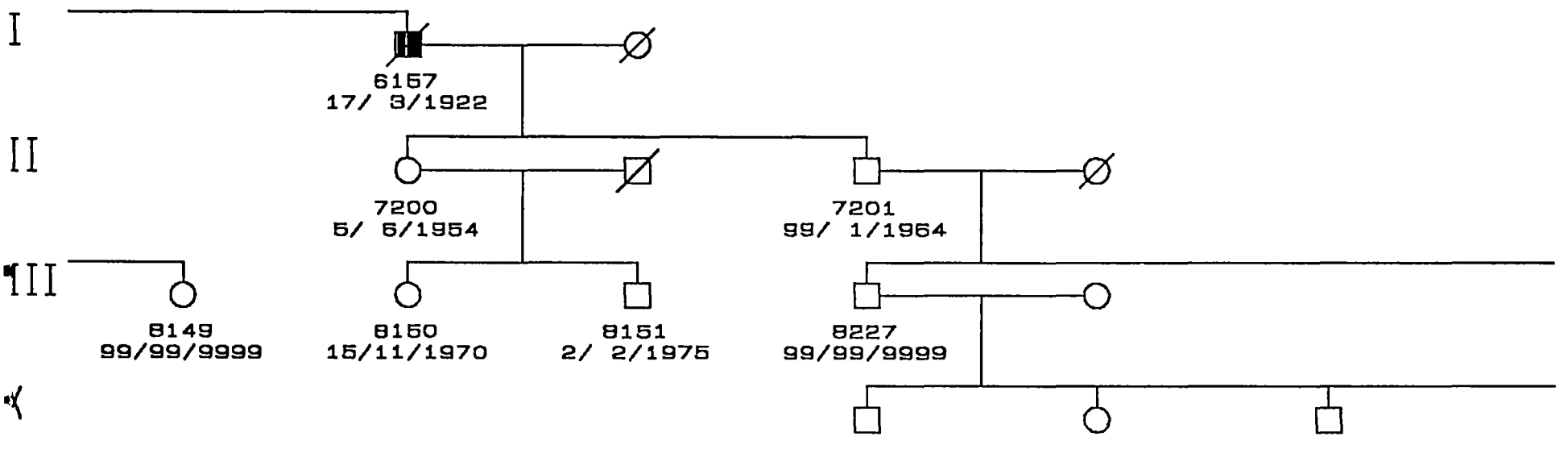
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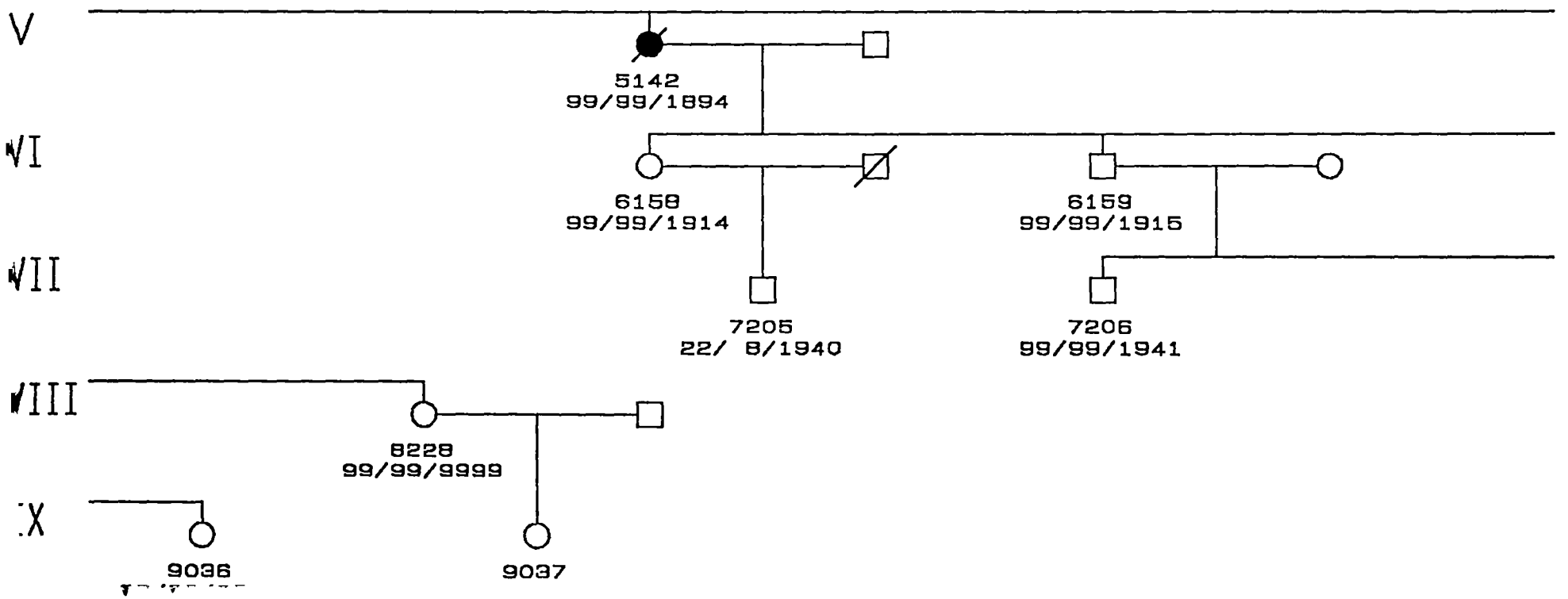
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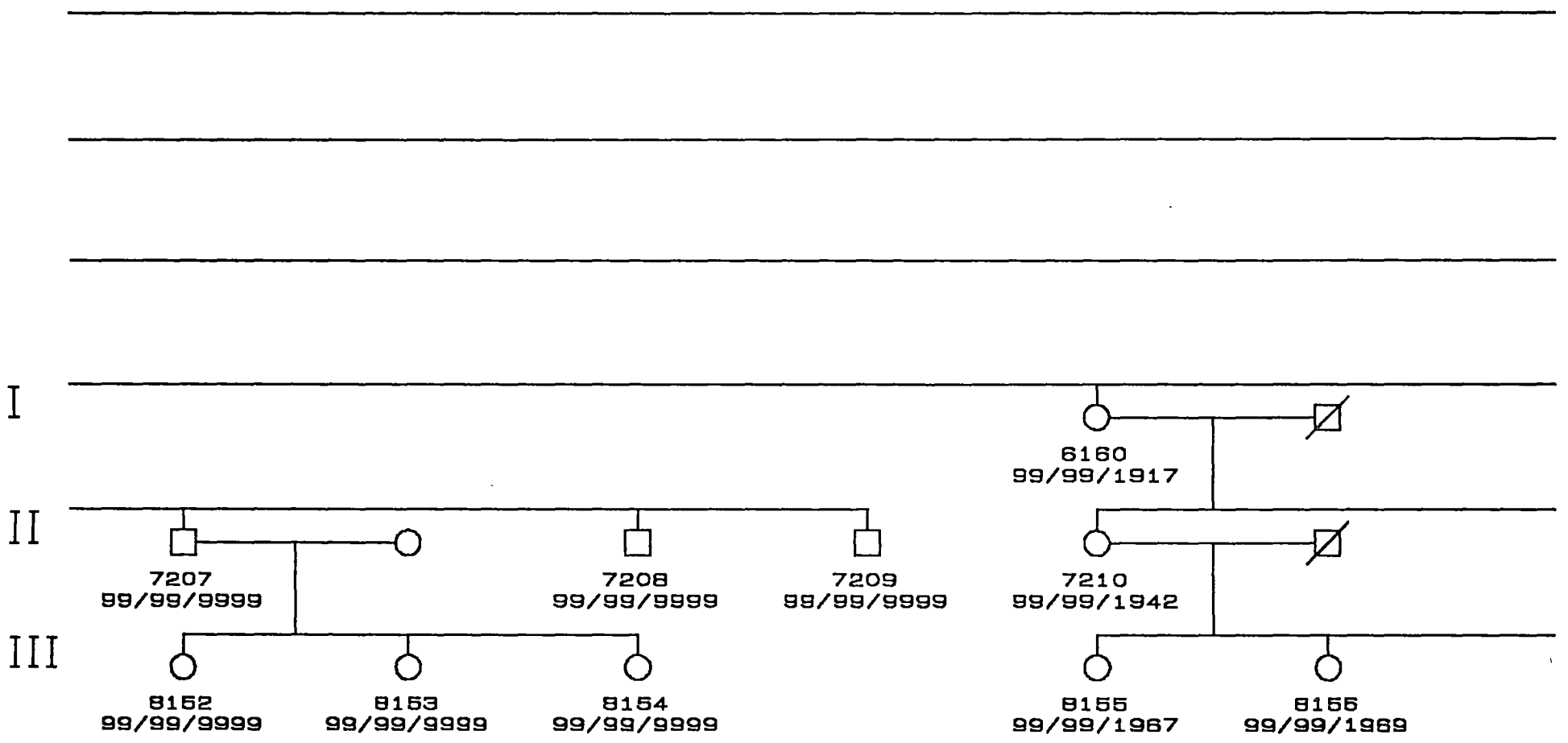
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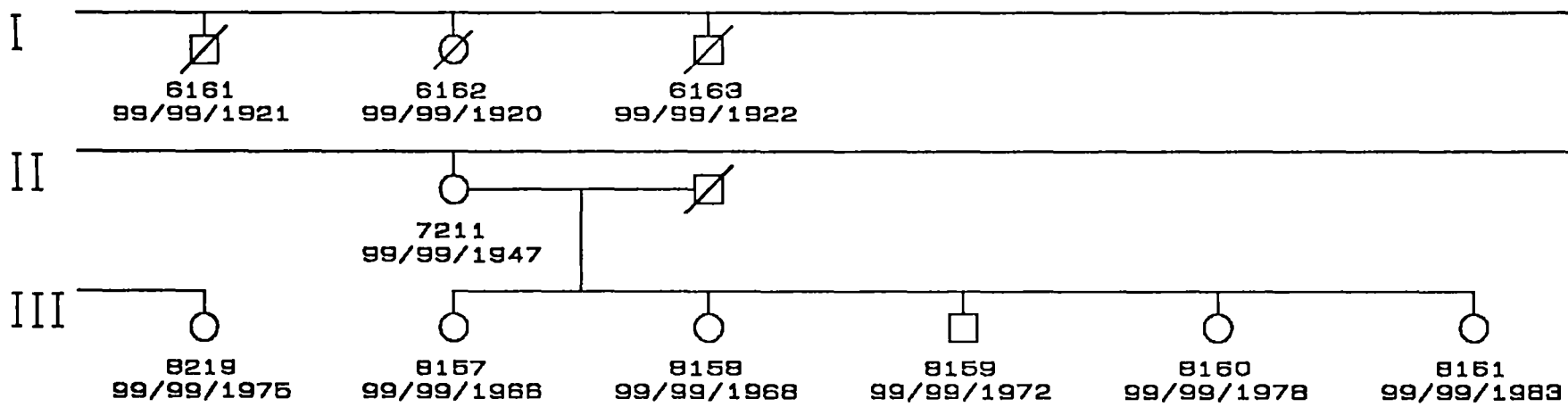
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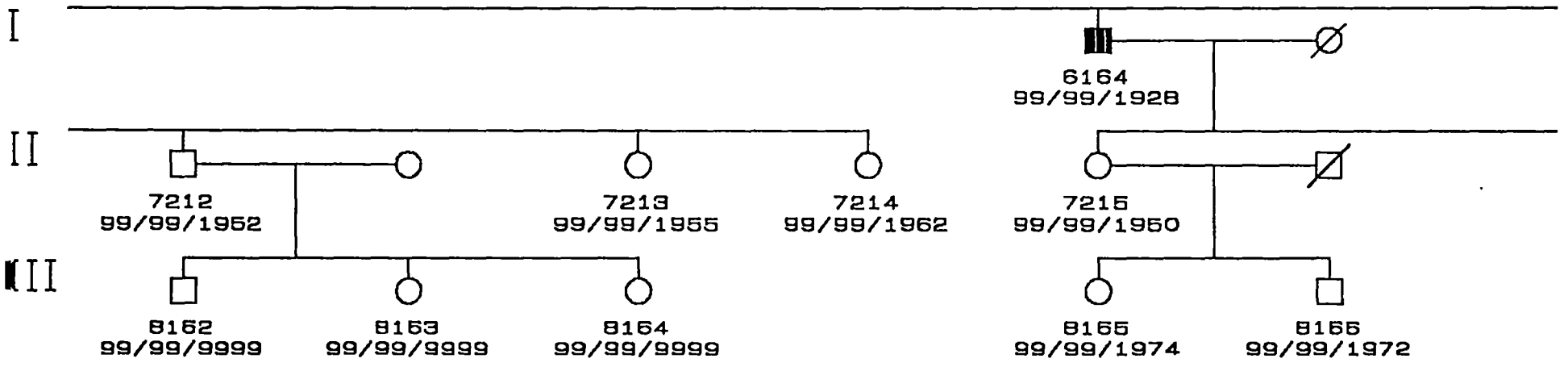
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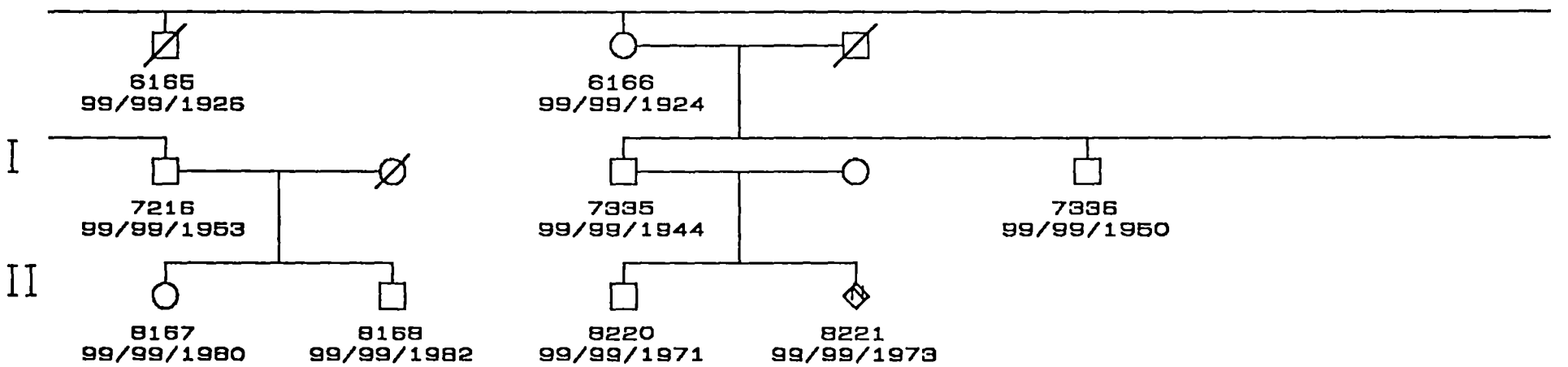
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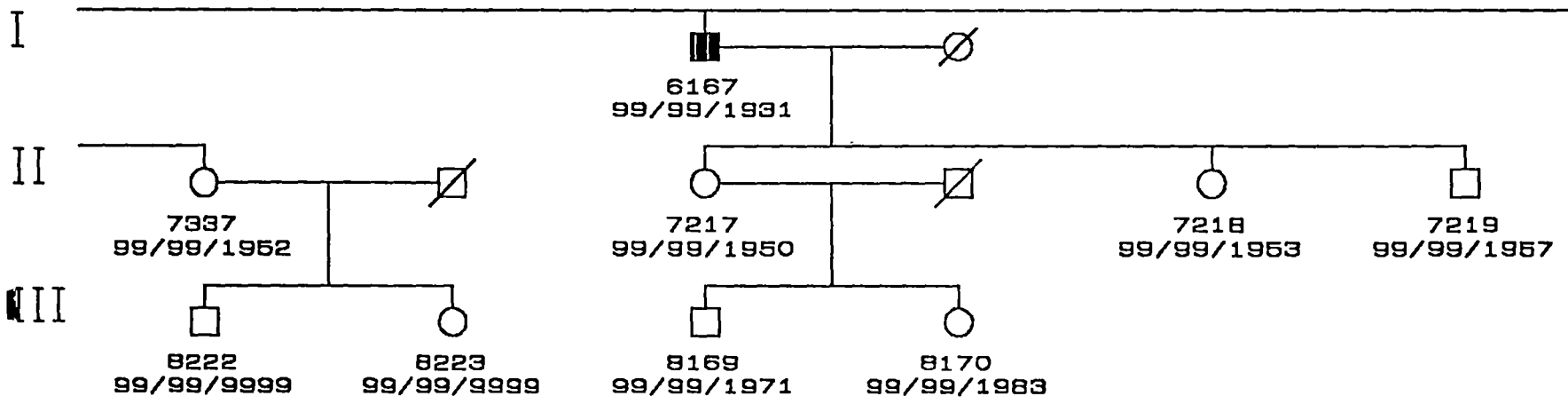
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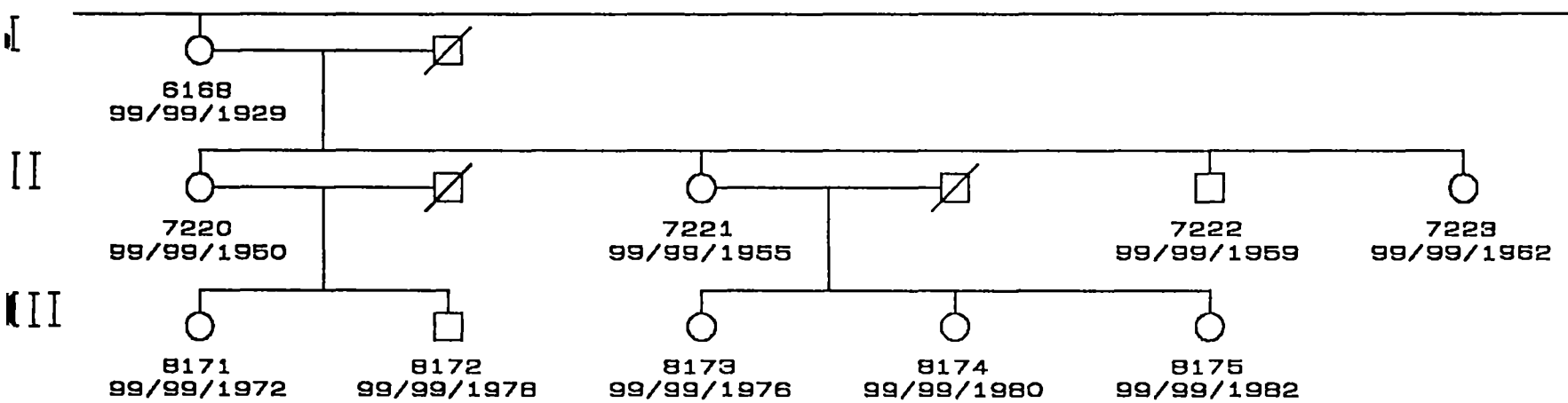
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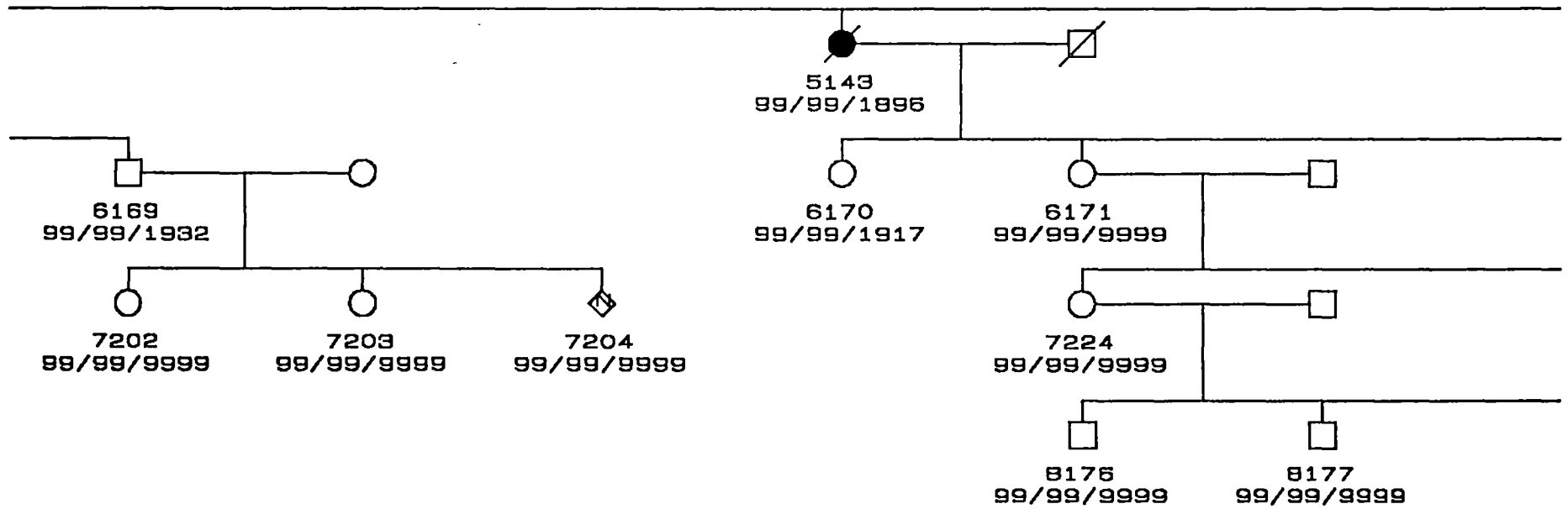
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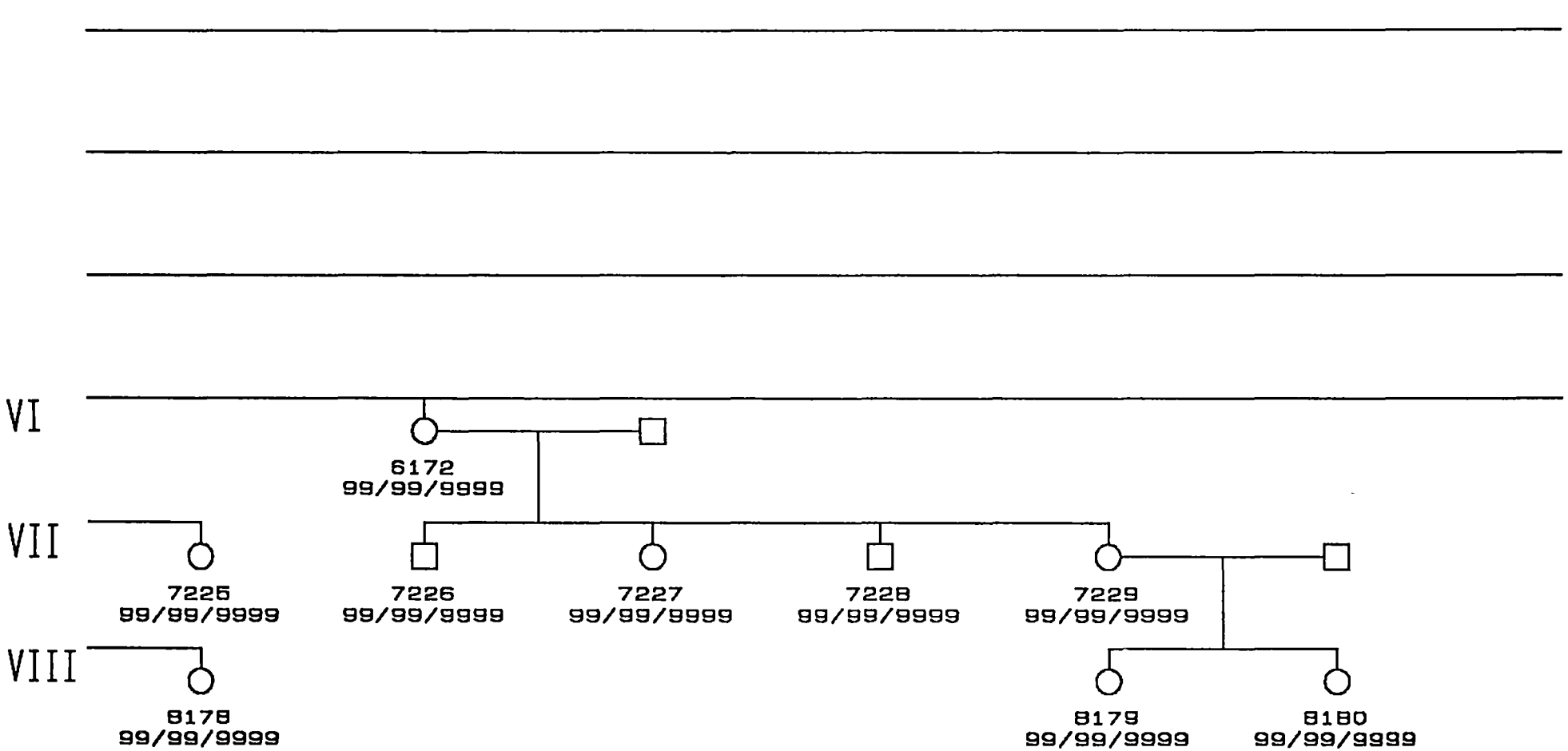
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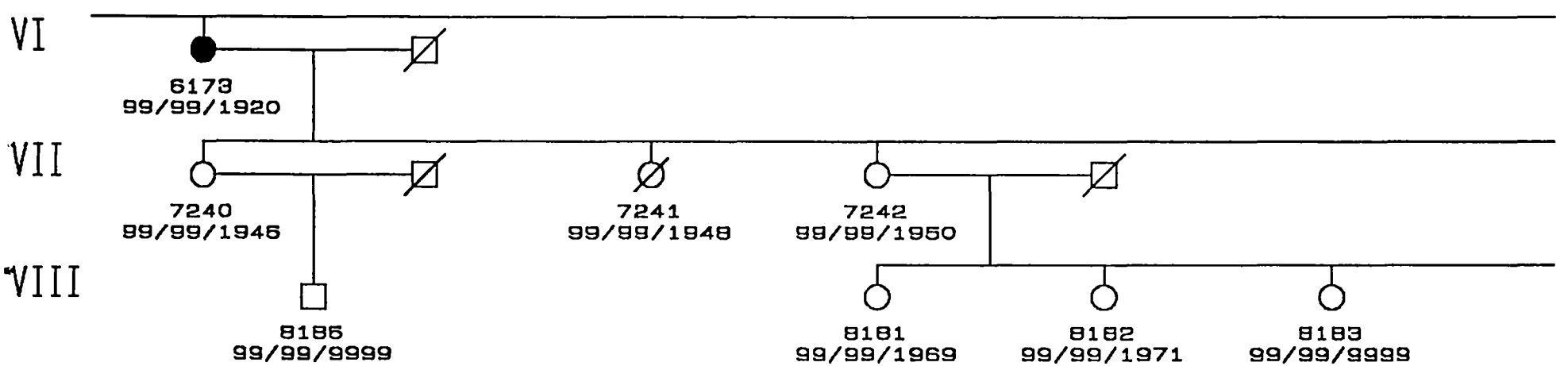
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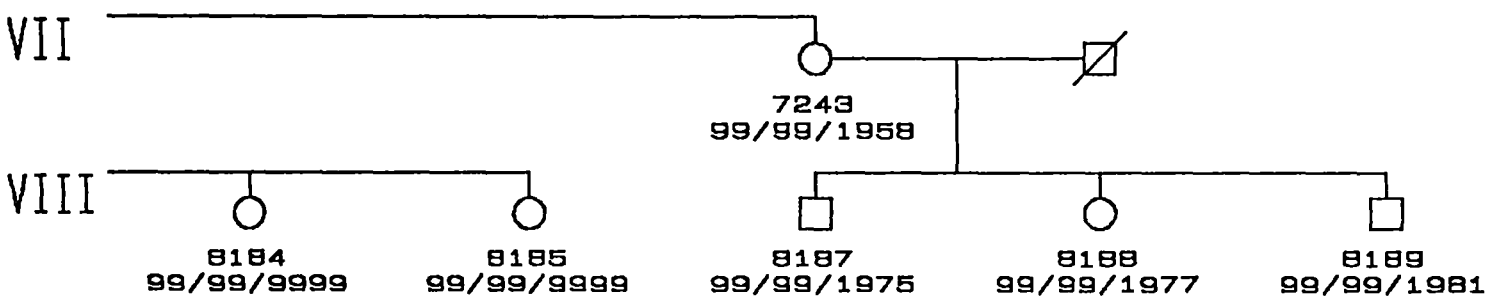
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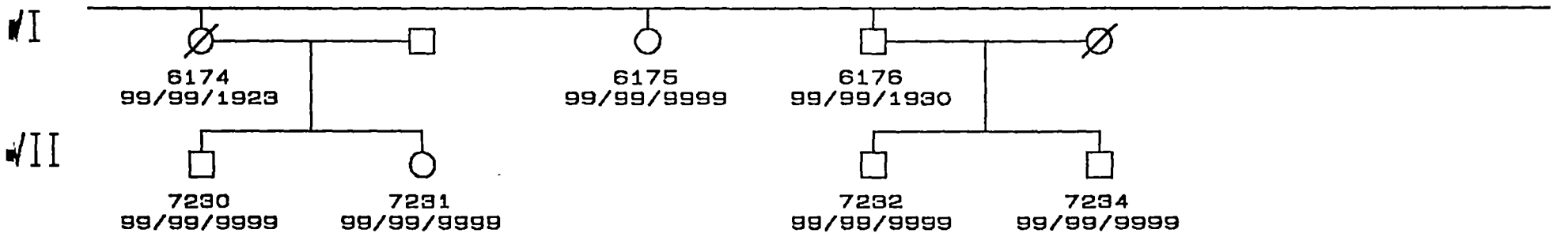
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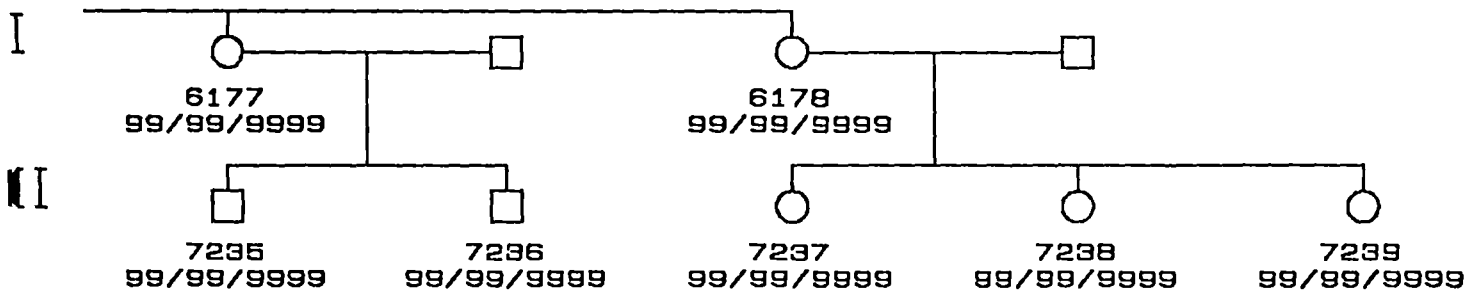
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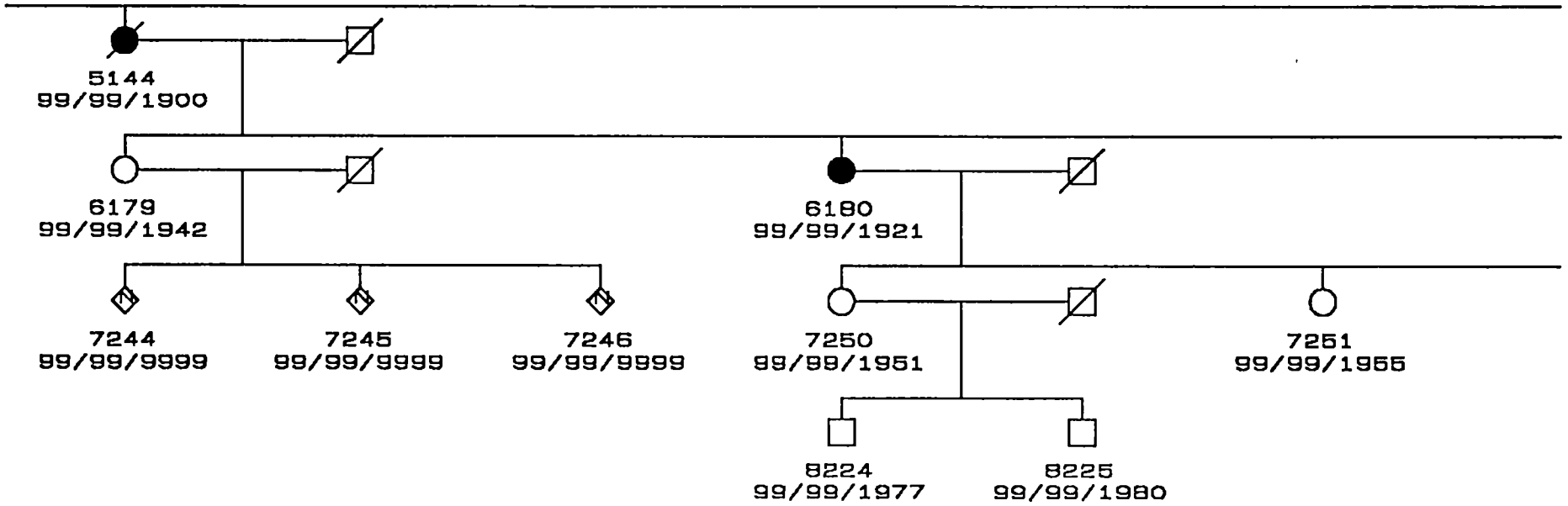
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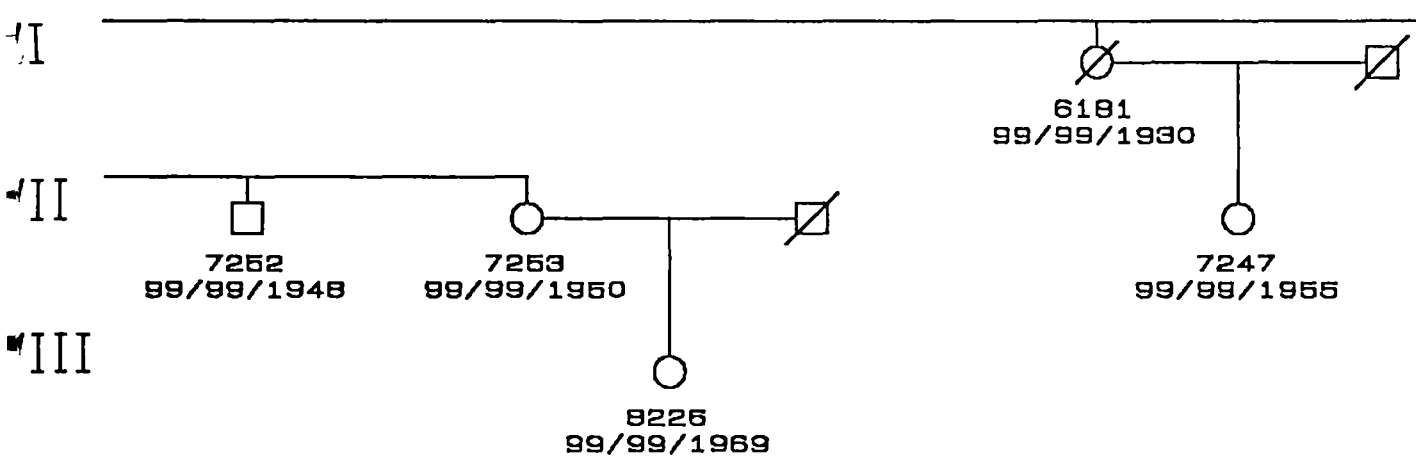
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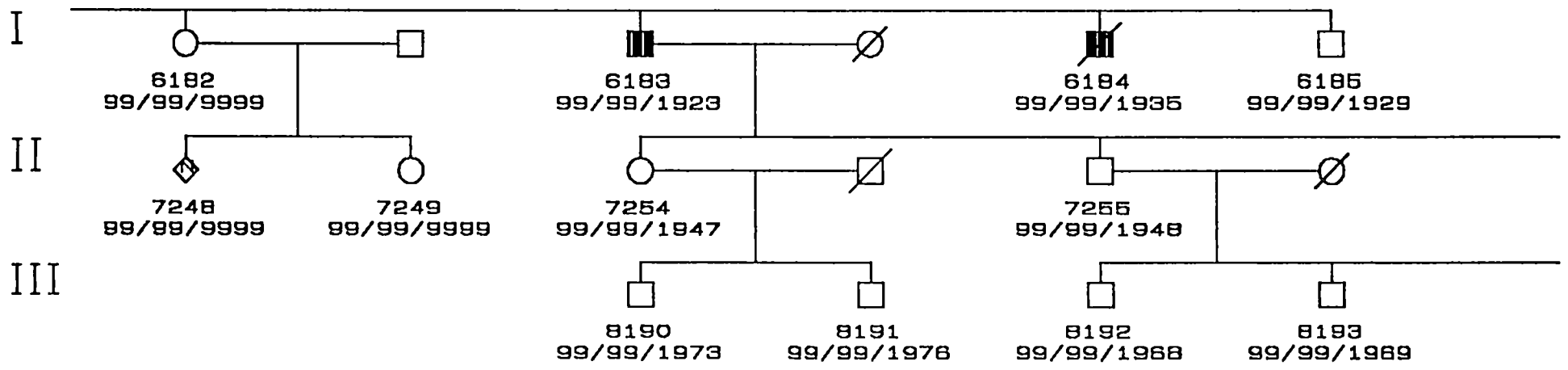
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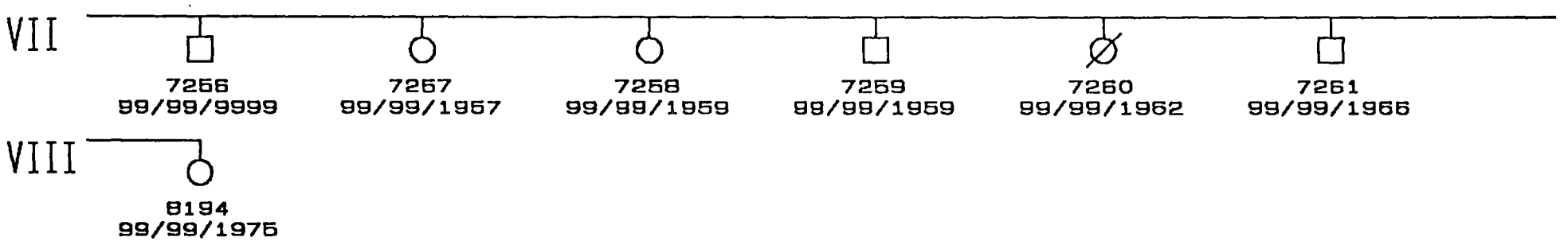
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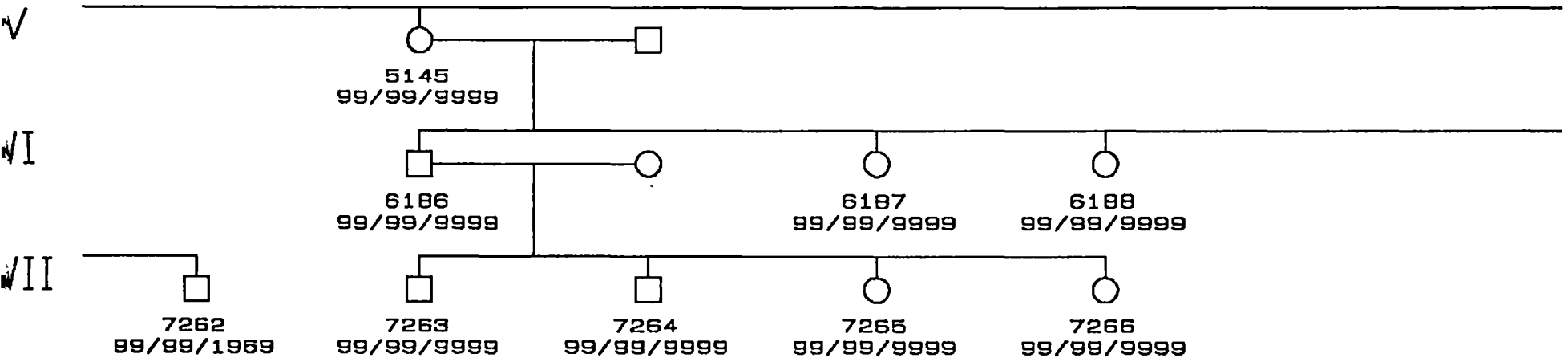
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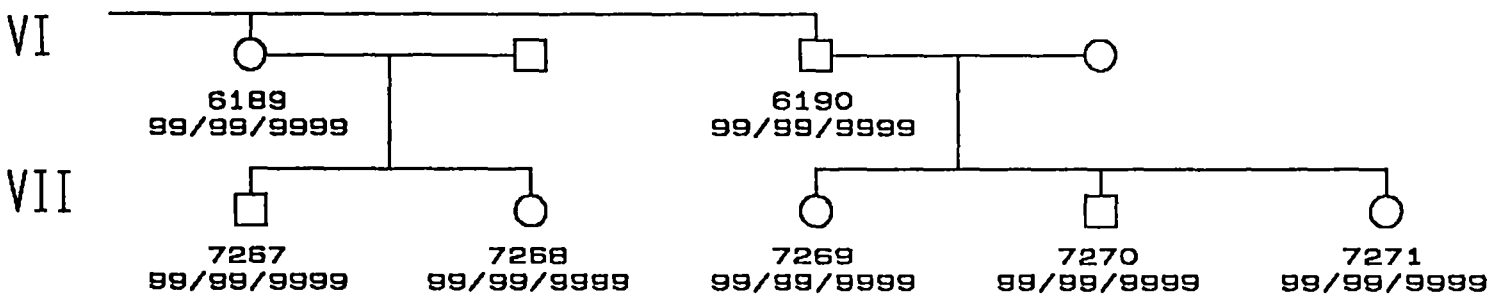
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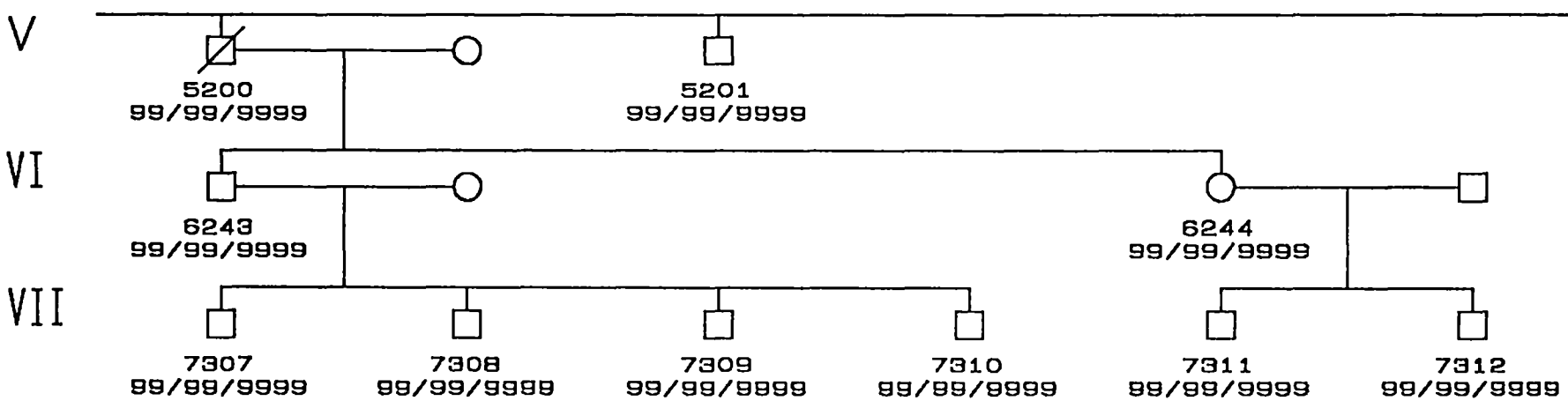
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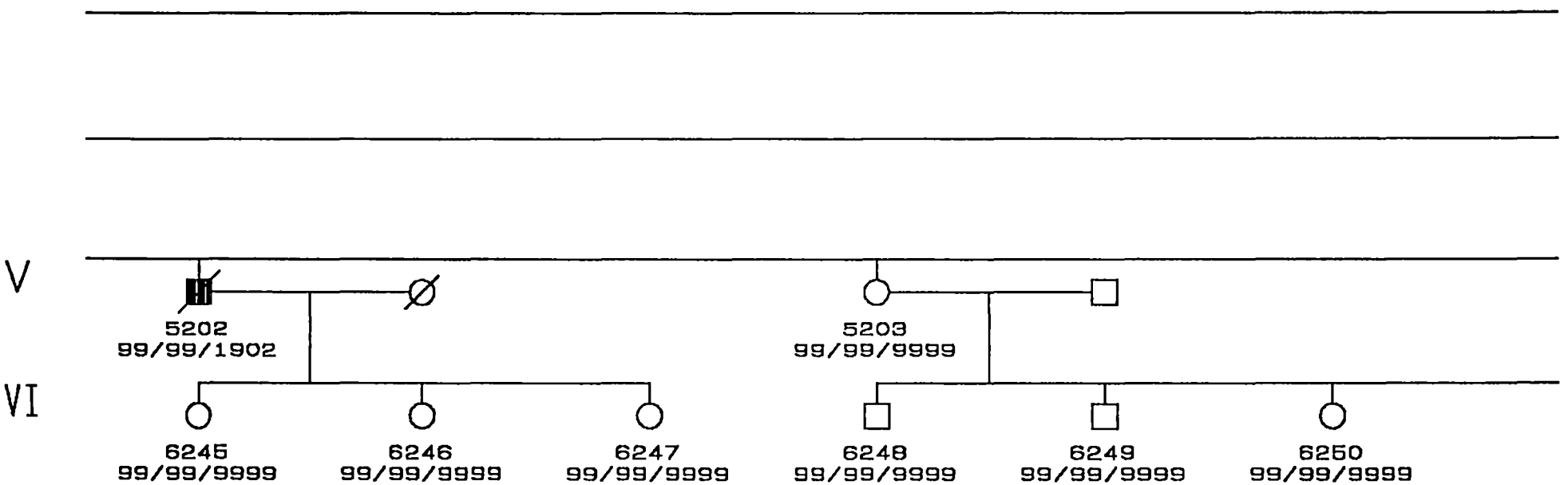
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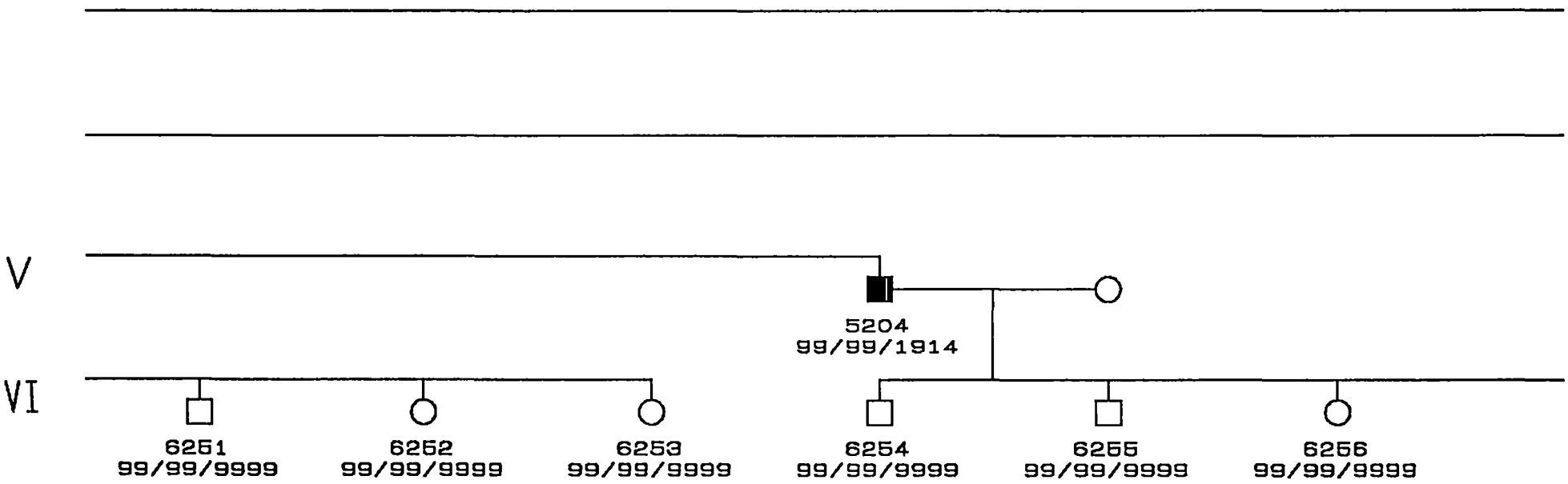
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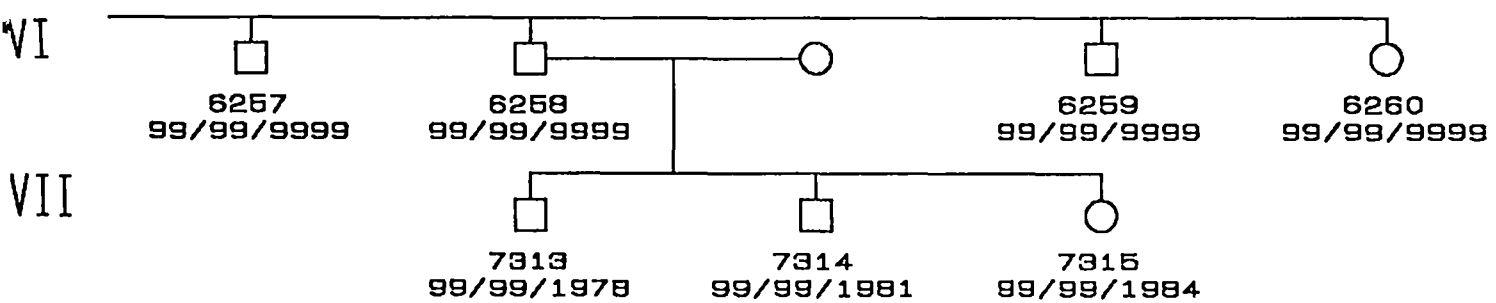
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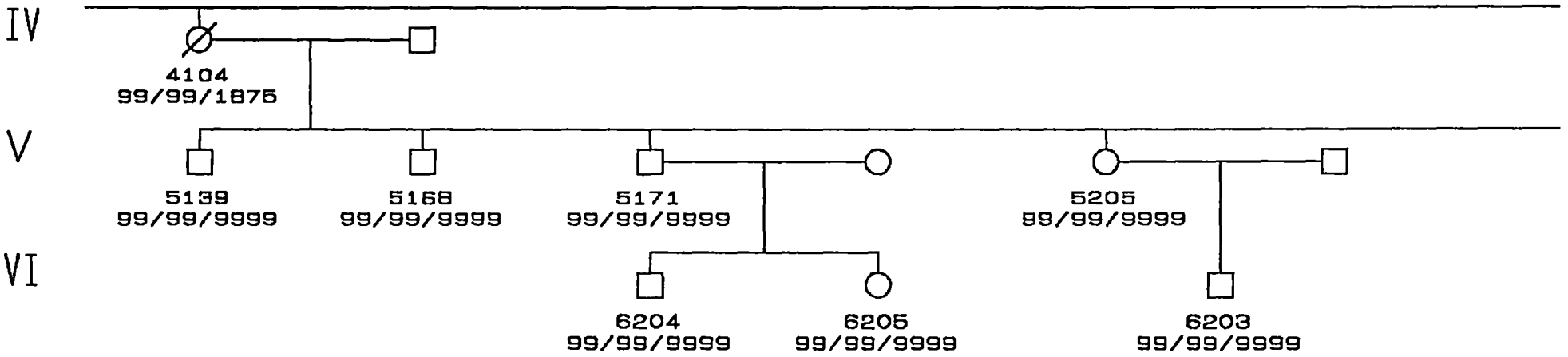
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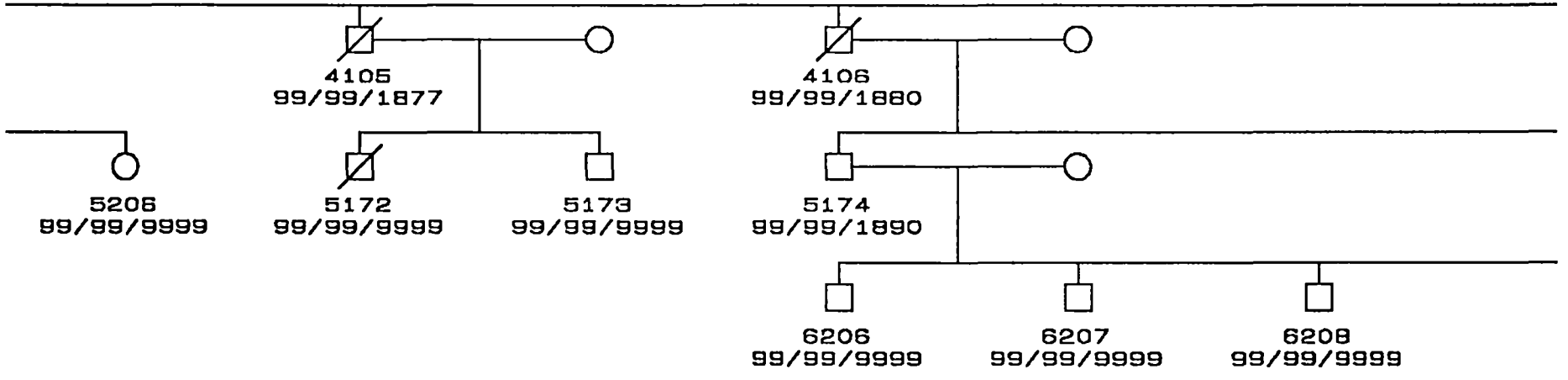
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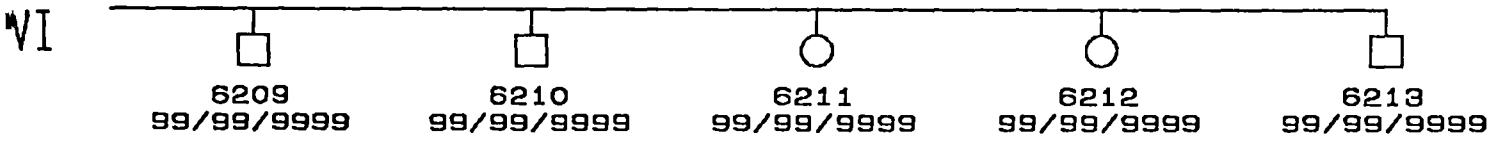
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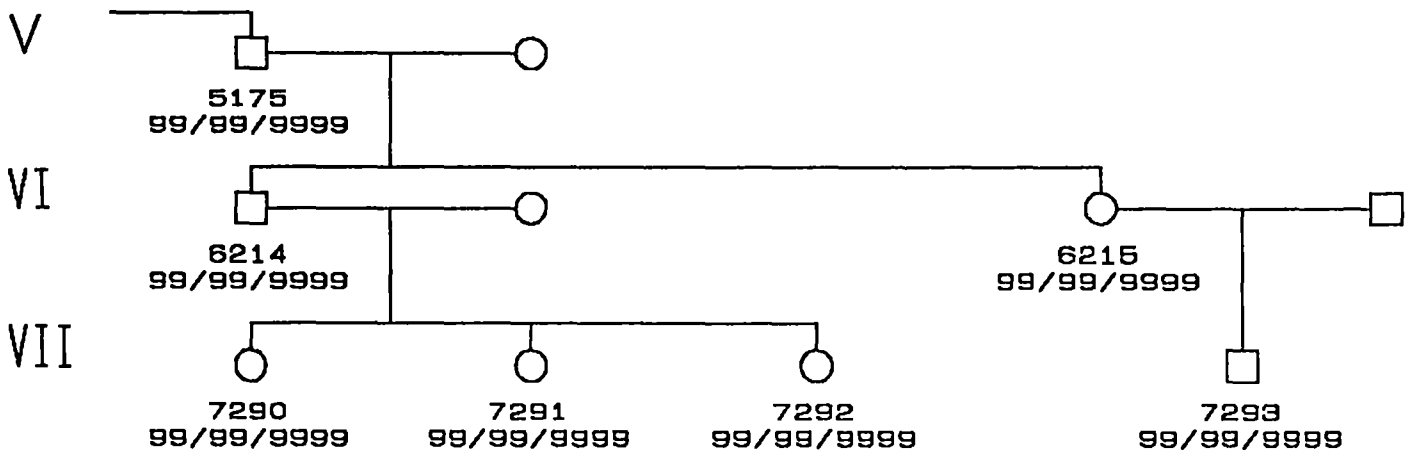
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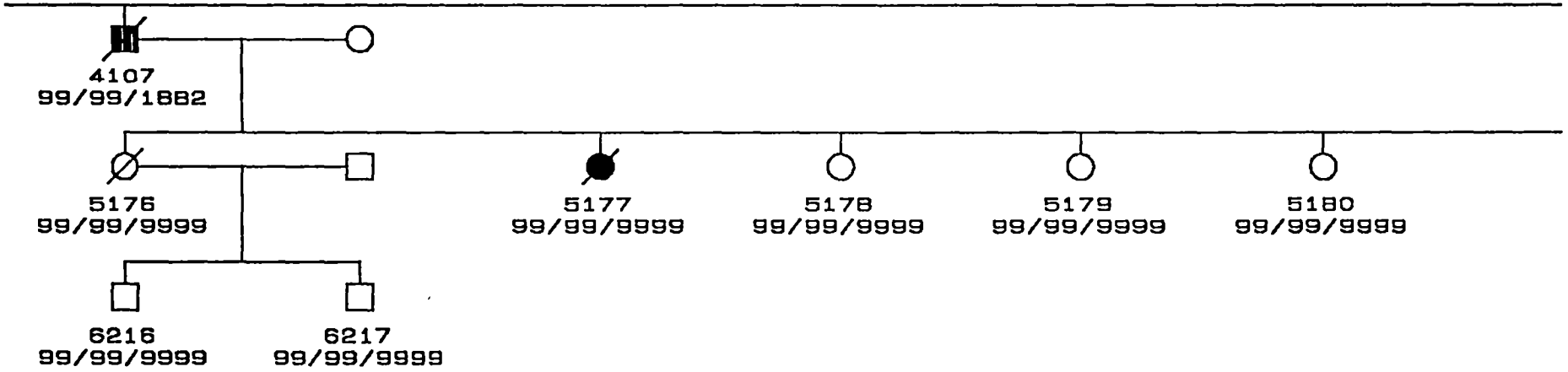
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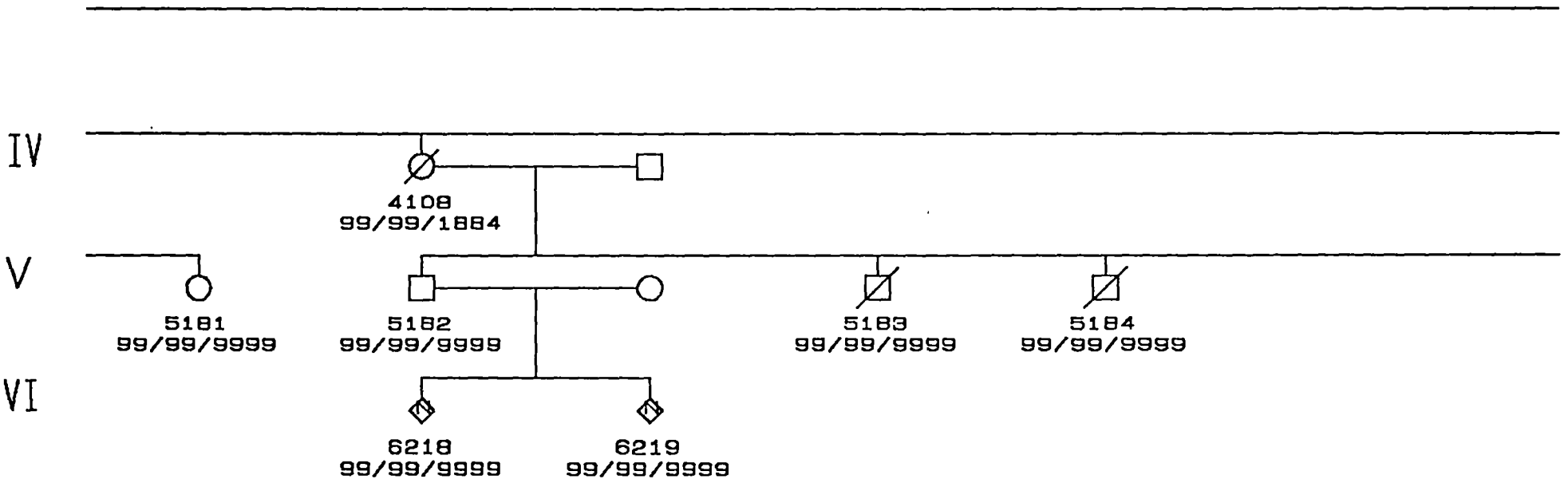
5180
99/99/9999
6216
99/99/9999
6217
99/99/9999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VI
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5181
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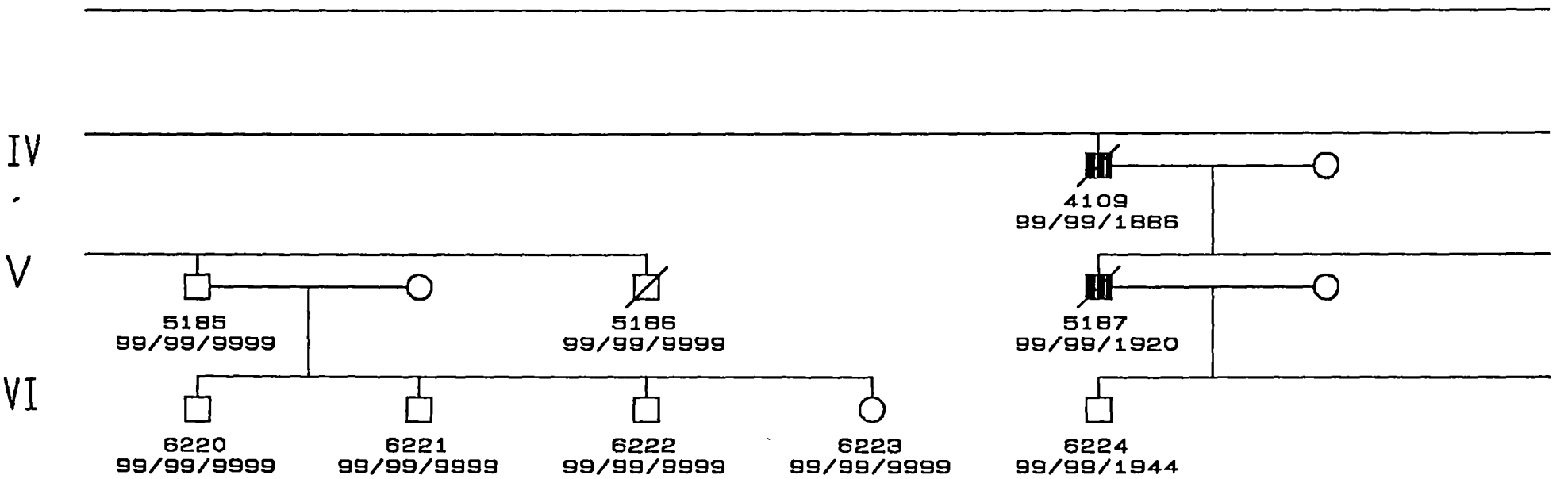
99/99/9999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V
VI
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99/99/1920
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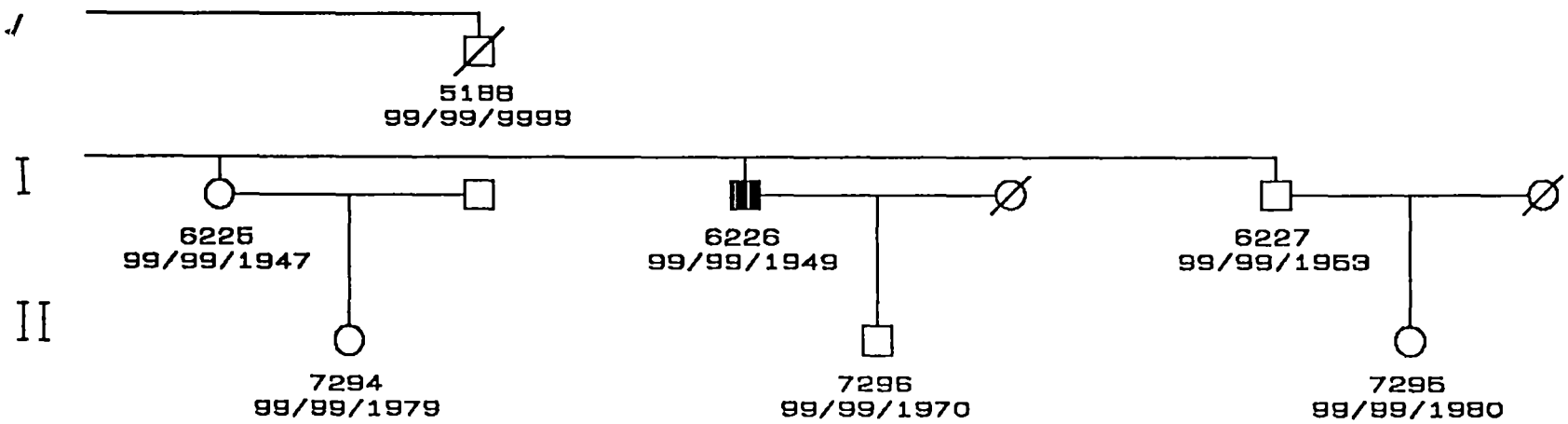
99/99/1944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II
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6225
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7294
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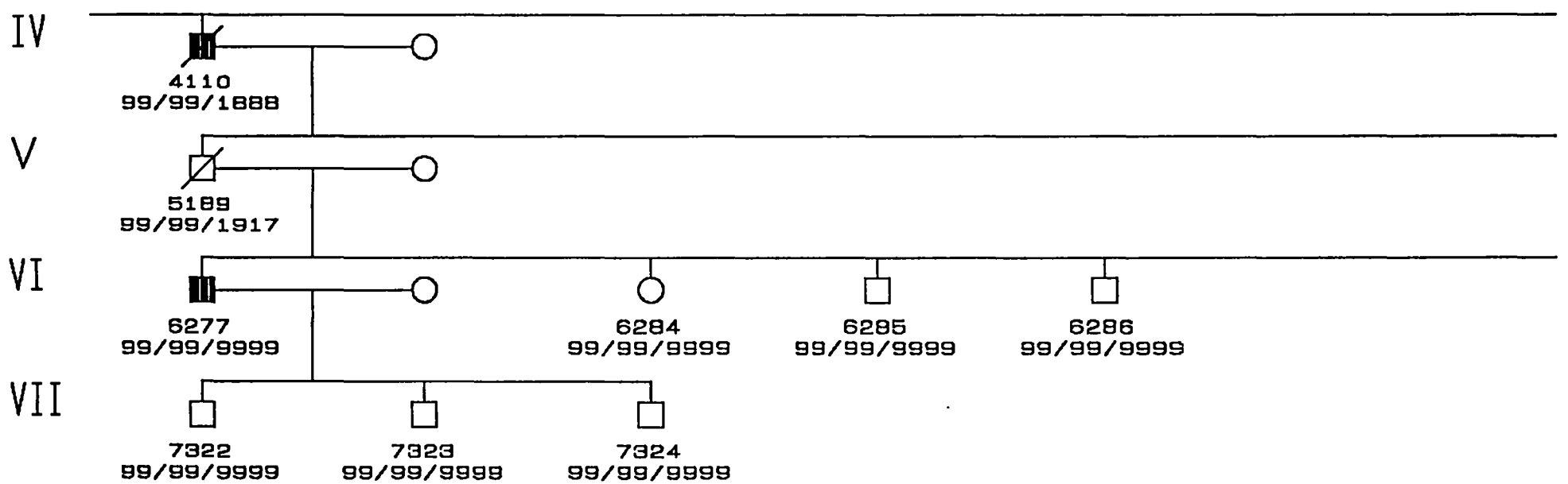
7296
99/99/1970
7295
99/99/1980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V
VI
VII
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5189
99/99/1917
6277
99/99/9999
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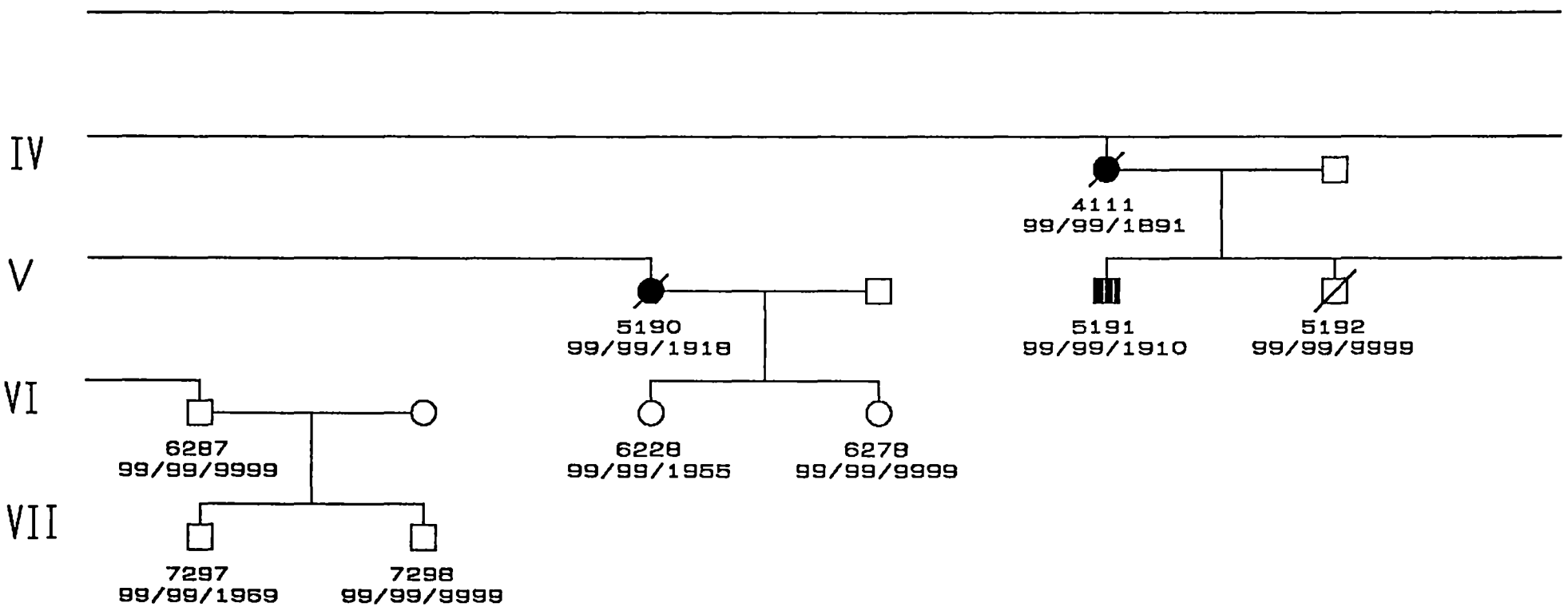
7323
99/99/9999
7324
99/99/9999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V
VI
VII
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99/99/1891
5190
99/99/1918
5191
99/99/1910
5192
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6278
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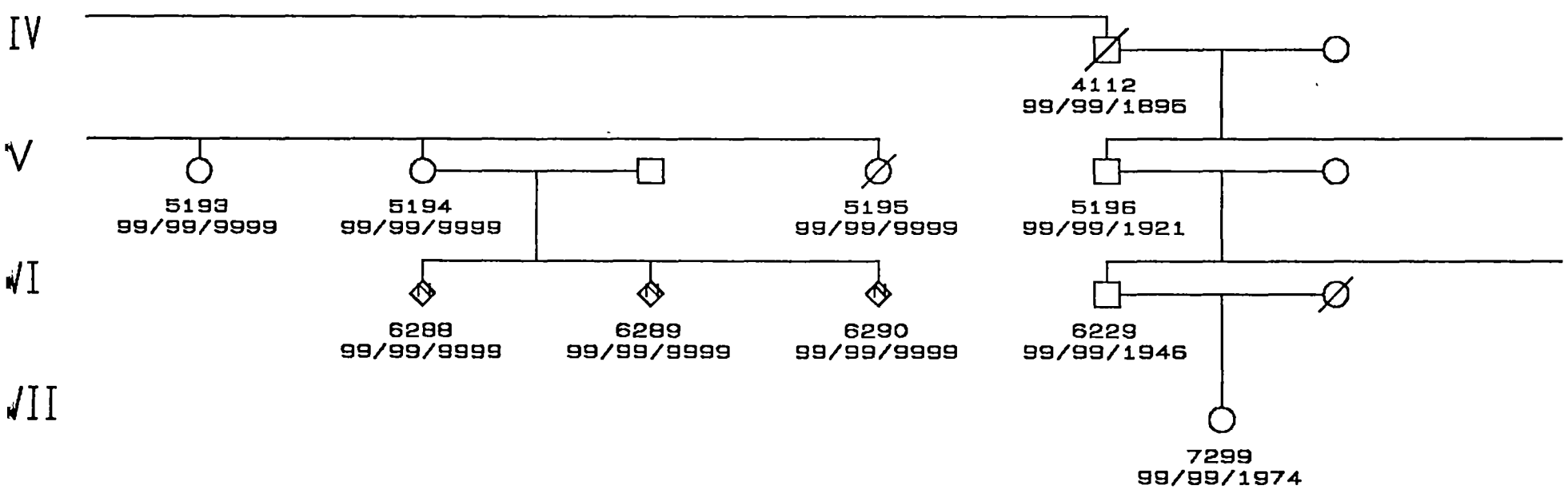
99/99/1969
7298
99/99/9999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V
VI
VII
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5195
99/99/9999
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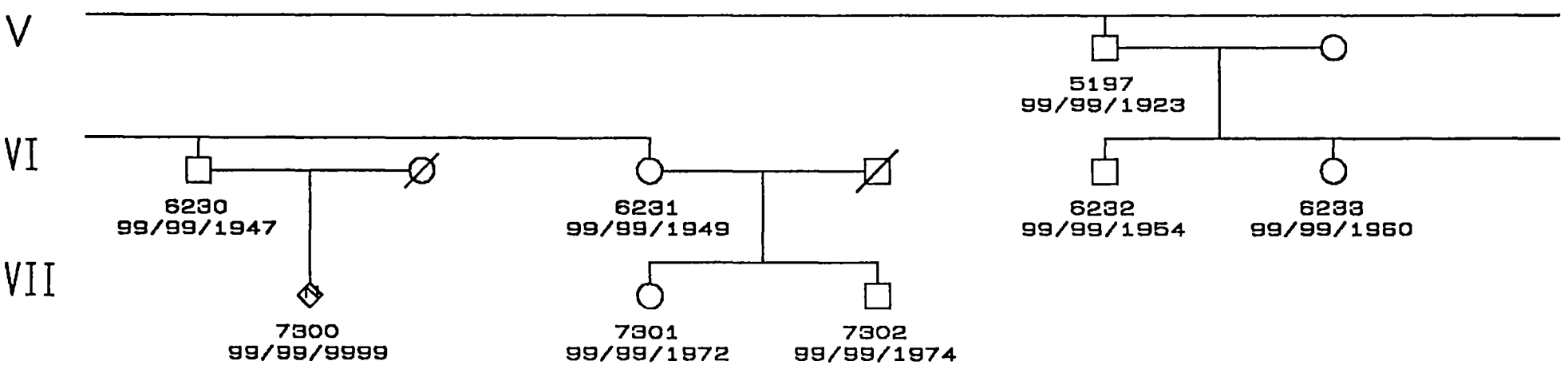
7299
99/99/1974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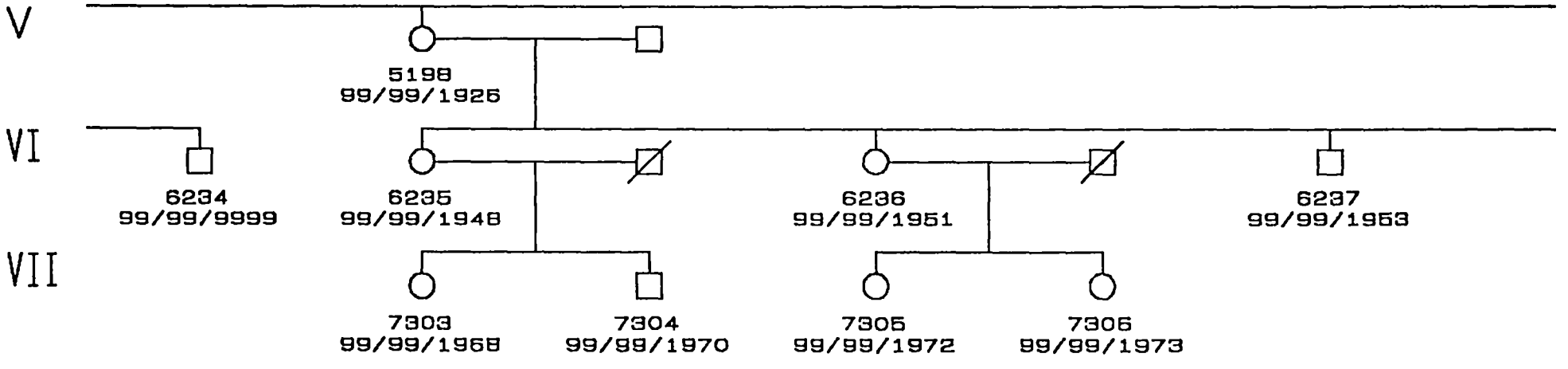
VI
VII
5197
99/99/1923
6230
99/99/1947
6231
99/99/1949
6232
99/99/1954
6233
99/99/1960
7300
99/99/9999
7301
99/99/1972
7302
99/99/1974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VI
VII
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99/99/1970
7305
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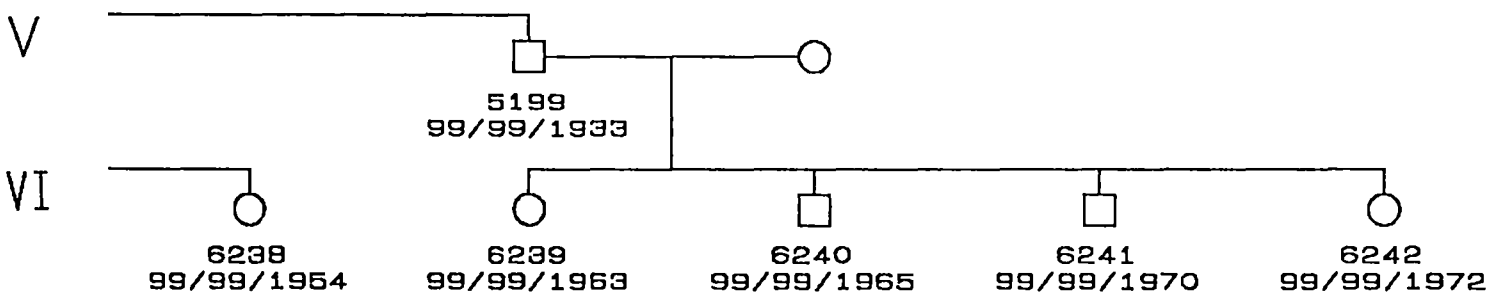
7306
99/99/1973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VI
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6240
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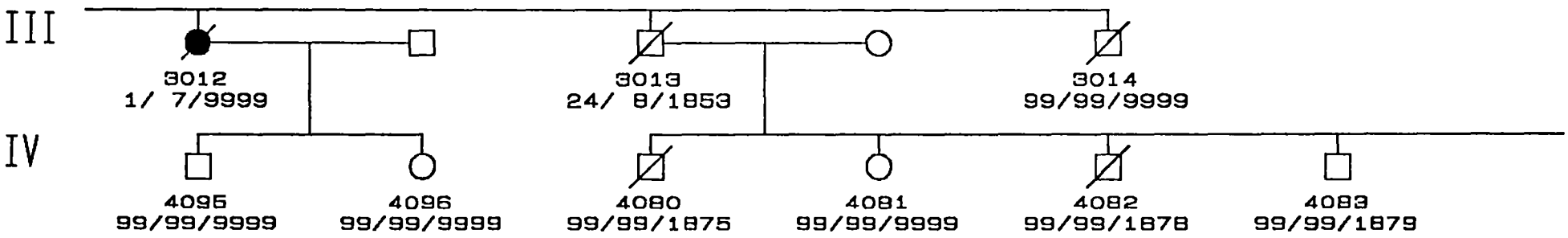
99/99/1970
6242
99/99/1972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IV
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1/ 7/9999
3013
24/ 8/1853
3014
99/99/9999
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99/99/9999
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4080
99/99/1875
4081
99/99/9999
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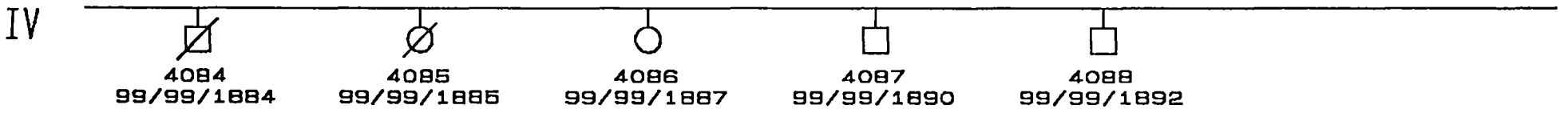
99/99/1878
4083
99/99/1879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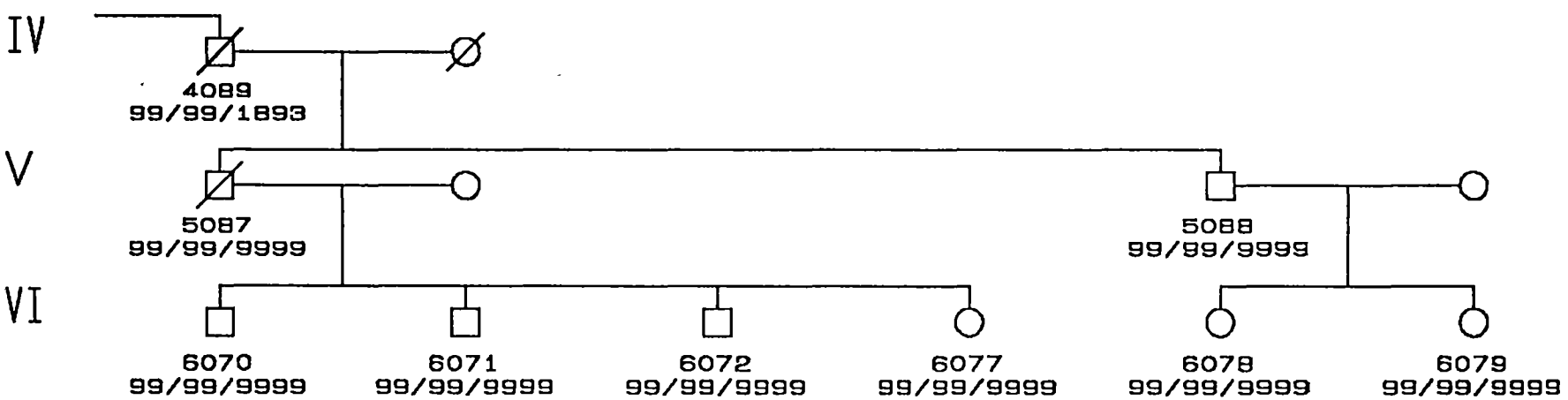
99/99/1885
4086
99/99/1887
4087
99/99/1890
4088
99/99/1892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V
VI
4089
99/99/1893
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99/99/9999
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99/99/9999
6071
99/99/9999
6072
99/99/9999
6077
99/99/9999
6078
99/99/9999
6079
99/99/9999

DEPARTMENT OF CLINICAL NEUROPHYSIOLOGY

CADWELL SPECTRUM 32

Patient Clinical Record

12/2/88

Neurometric Analysis (QEEG) Discriminants

Classification functions in this program were derived from groups of patients who met specific criteria for group membership.

Neurometric classifications are statistical in nature and serve only as an adjunct to the other clinical results used to evaluate the patient.

This patient's discriminant scores lie outside of the normal limits expected for an individual of this age.

ne: GK F
e: 27.4 yrs

Analyzed: 05/30/90 Recorded: 12/02/88
Epochs: 38

nopolar Raw Measures

		Fp1	Fp2	F7	F8	F3	F4	C3	C4	Fpz	Fz	Cz
bsolute	Δ	7.8	7.4	5.2	6.1	13.5	11.1	16.1	12.0	7.9	15.2	20.4
Power	θ	5.7	5.3	4.4	3.9	8.1	7.2	7.0	5.9	5.8	8.8	8.6
(uV²)	α	1.7	1.7	1.5	1.6	1.9	2.0	1.7	1.5	1.7	2.0	1.9
	ß	2.7	3.3	3.0	2.8	3.0	3.6	2.6	3.4	2.6	3.1	2.9
	T	17.8	17.6	14.1	14.3	26.5	23.8	27.4	22.8	18.1	29.1	33.8
elative	Δ	43.7	42.1	36.9	42.4	51.0	46.4	58.8	52.5	43.9	52.3	60.4
Power	θ	32.0	29.9	31.1	27.4	30.5	30.0	25.7	25.7	32.2	30.1	25.4
(%)	α	9.4	9.4	10.9	10.8	7.2	8.4	6.0	6.8	9.4	7.0	5.6
	ß	14.9	18.6	21.1	19.3	11.2	15.1	9.5	15.0	14.5	10.6	8.6

Power	Δ	2.5	-7.7	10.0	14.9
ymmetry	θ	4.0	5.6	6.1	9.2
(%)	α	0.8	-0.4	-2.3	3.7
	ß	-10.6	3.5	-9.3	-13.7
herence	Δ	88.6	28.0	88.1	87.7
(%)	θ	89.9	41.1	83.1	78.9
	α	86.2	42.8	77.4	61.3
	ß	74.9	31.1	66.8	57.6

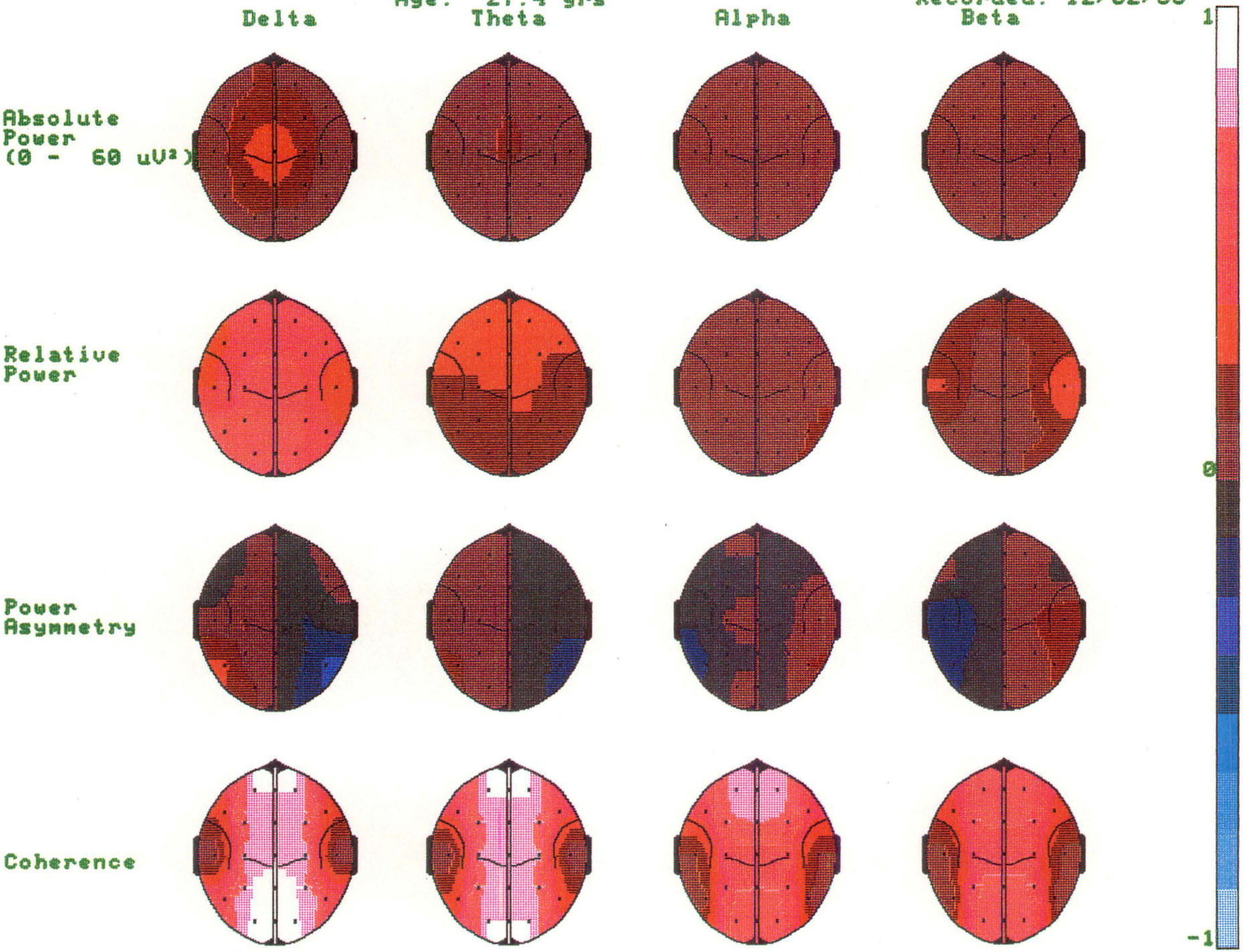
		T3	T4	T5	T6	P3	P4	O1	O2	Pz	Oz
bsolute	Δ	4.4	4.1	5.4	3.0	13.2	10.9	7.1	5.3	16.6	5.7
Power	θ	2.8	2.4	2.4	1.7	5.6	5.1	3.0	2.5	7.3	2.7
(uV²)	α	1.2	1.6	0.8	1.1	1.4	1.5	1.4	1.3	1.7	1.2
	ß	3.1	5.2	1.0	1.5	1.9	2.2	1.3	1.4	2.3	1.3
	T	11.6	13.2	9.7	7.3	22.1	19.6	12.7	10.5	27.9	11.0
elative	Δ	38.1	30.9	56.2	40.8	59.8	55.5	55.6	50.9	59.5	52.3
Power	θ	24.2	18.0	24.6	23.8	25.2	26.0	23.2	23.4	26.1	24.7
(%)	α	10.7	11.9	8.4	14.8	6.3	7.4	11.1	12.5	5.9	11.0
	ß	27.0	39.2	10.8	20.6	8.7	11.0	10.1	13.2	8.4	12.0

Power	Δ	4.1	29.4	9.7	14.1
ymmetry	θ	8.4	15.8	4.4	9.3
(%)	α	-11.2	-14.2	-2.0	3.8
	ß	-24.4	-17.6	-5.6	-3.6
herence	Δ	4.3	55.2	92.9	93.6
(%)	θ	0.9	30.1	86.1	88.3
	α	11.9	7.2	61.2	75.5
	ß	7.3	2.6	69.8	75.1

Monopolar Raw Maps

Name: GK F
Age: 27.4 yrs

Analyzed: 05/30/90
Recorded: 12/02/88



ame: GK F
ge: 27.4 yrs

Analyzed: 05/30/90 Recorded: 12/02/88
Epochs: 38

opolar Normative Measures

		Fp1	Fp2	F7	F8	F3	F4	C3	C4	Fpz	Fz	Cz
solute	Δ	10.0	9.7	6.5	6.0	10.8	10.3	10.5	10.7	9.8	12.8	15.8
ower	θ	6.8	6.6	5.0	4.7	9.8	9.4	9.9	10.0	6.7	12.6	15.6
uU²)	α	8.8	8.6	6.6	6.4	12.4	12.1	14.5	15.6	8.7	15.2	20.7
	β	4.5	4.3	3.6	3.4	5.9	5.9	6.3	6.4	4.4	6.5	8.2
lative	Δ	32.1	32.0	28.7	28.3	26.2	26.0	24.3	24.1	32.1	25.9	25.1
ower	θ	21.3	21.4	22.0	21.8	24.1	23.8	22.6	22.3	21.4	25.6	24.5
(%)	α	28.5	28.8	29.9	31.0	31.3	31.5	34.7	35.4	28.6	31.7	33.4
	β	14.3	14.1	15.9	15.9	14.6	15.1	14.5	14.1	14.2	12.9	12.9

ower	Δ	1.7	3.3	2.1	-1.9
mmetry	θ	1.4	3.3	2.3	-1.7
(%)	α	0.9	1.1	1.3	-3.2
	β	1.8	2.6	0.3	-1.0
erence	Δ	87.4	29.1	83.6	82.2
(%)	θ	86.4	29.8	83.0	76.6
	α	92.7	52.8	88.2	68.0
	β	78.6	28.9	73.1	54.6

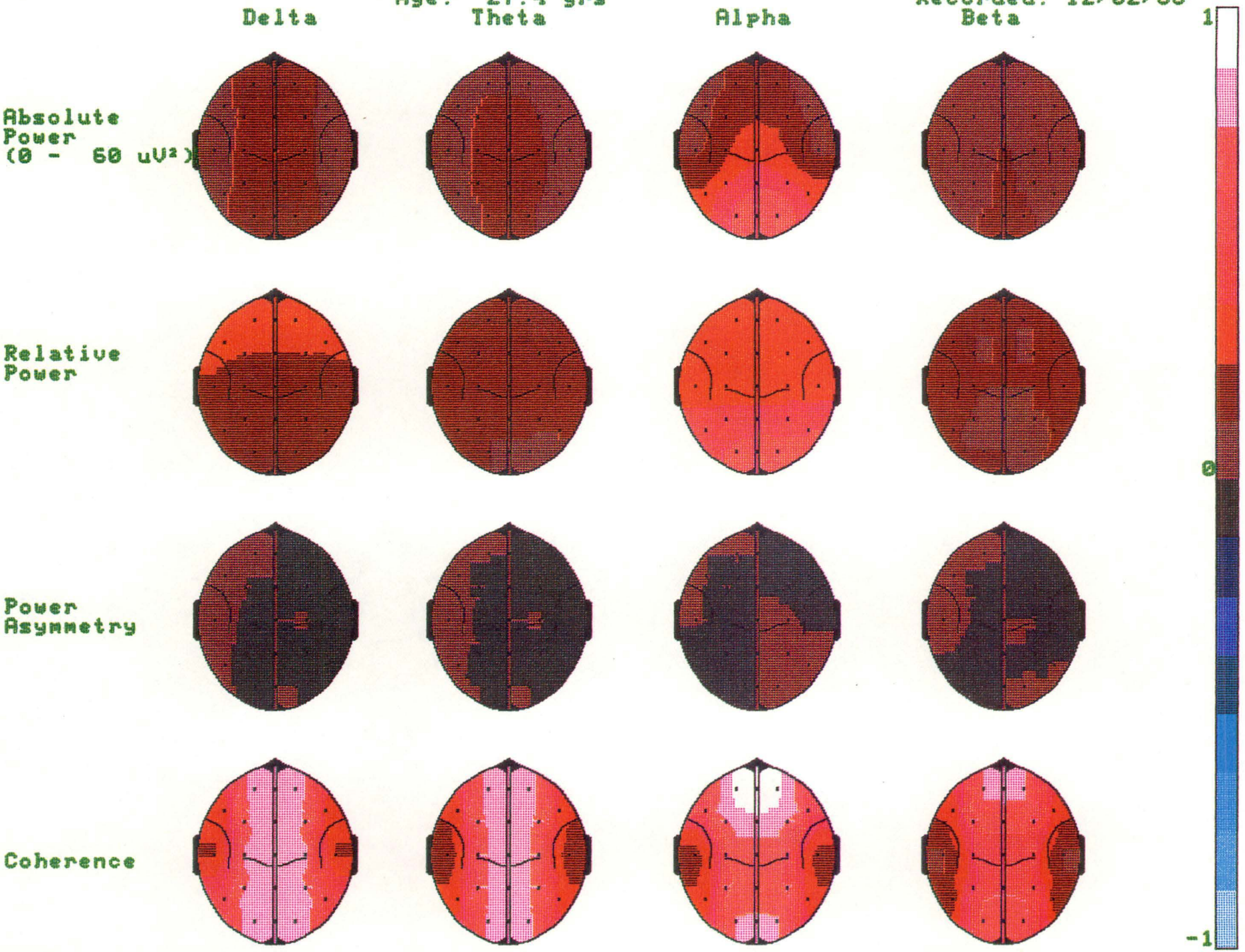
		T3	T4	T5	T6	P3	P4	O1	O2	Pz	Oz
solute	Δ	4.8	4.3	7.2	6.9	11.4	11.2	9.5	9.7	14.0	9.6
ower	θ	4.2	3.7	6.5	6.1	10.0	9.8	8.2	8.2	12.7	8.2
uU²)	α	6.3	6.1	17.0	20.5	25.6	27.0	33.2	38.1	31.7	35.6
	β	4.0	3.5	6.0	6.1	8.0	8.1	8.1	8.6	8.8	8.3
elative	Δ	23.6	22.3	18.6	16.6	19.4	18.6	15.2	13.9	19.4	14.5
ower	θ	20.5	19.1	16.4	14.1	16.9	16.2	13.0	12.0	17.3	12.5
(%)	α	32.1	32.5	45.4	50.5	45.5	47.2	54.9	57.4	46.4	56.1
	β	20.0	18.5	15.3	14.5	13.4	13.2	12.8	12.4	12.2	12.6

Power	Δ	5.2	1.7	0.4	-0.4
mmetry	θ	5.8	3.4	0.7	-0.5
(%)	α	2.0	-8.8	-3.5	-6.9
	β	6.7	-1.0	-0.7	-2.7
herence	Δ	25.1	55.2	85.7	86.7
(%)	θ	13.5	32.4	78.4	81.1
	α	18.0	29.6	70.3	77.5
	β	10.4	17.3	61.4	70.1

Monopolar Normative Maps

Name: GK F
Age: 27.4 yrs

Analyzed: 05/30/90
Recorded: 12/02/88



e: GK F
e: 27.4 yrs

Analyzed: 05/30/90 Recorded: 12/02/88
Epochs: 38

ıopolar Z Score Measures

		Fp1	Fp2	F7	F8	F3	F4	C3	C4	Fpz	Fz	Cz
bsolute	Δ	-0.47	-0.52	-0.51	0.03	0.58	0.16	1.05	0.26	-0.42	0.42	0.61
Power	θ	-0.36	-0.48	-0.29	-0.35	-0.45	-0.62	-0.74	-1.14	-0.29	-0.80	-1.33
	α	-2.16	-2.15	-1.93	-1.80	-2.42	-2.32	-2.73	-2.89	-2.13	-2.55	-3.01
	ß	-1.11	-0.61	-0.36	-0.39	-1.31	-0.93	-1.56	-1.14	-1.12	-1.36	-1.82
elative	Δ	0.91	0.82	0.79	1.31	2.16	1.80	2.82	2.35	0.94	2.23	2.79
Power	θ	1.71	1.38	1.48	1.04	0.93	0.92	0.44	0.51	1.73	0.63	0.14
	α	-2.12	-2.14	-2.14	-2.27	-2.84	-2.62	-3.43	-3.32	-2.13	-2.90	-3.31
	ß	0.11	0.78	0.80	0.57	-0.67	0.00	-1.03	0.16	0.06	-0.51	-0.98

Power	Δ	0.11	-0.65	1.05	2.25
ymmetry	θ	0.40	0.14	0.52	1.44
	α	-0.02	-0.10	-0.53	0.74
	ß	-1.53	0.06	-1.22	-1.41
herence	Δ	0.18	-0.06	0.85	1.02
	θ	0.67	0.53	0.02	0.37
	α	-1.09	-0.40	-1.33	-0.46
	ß	-0.32	0.16	-0.58	0.25

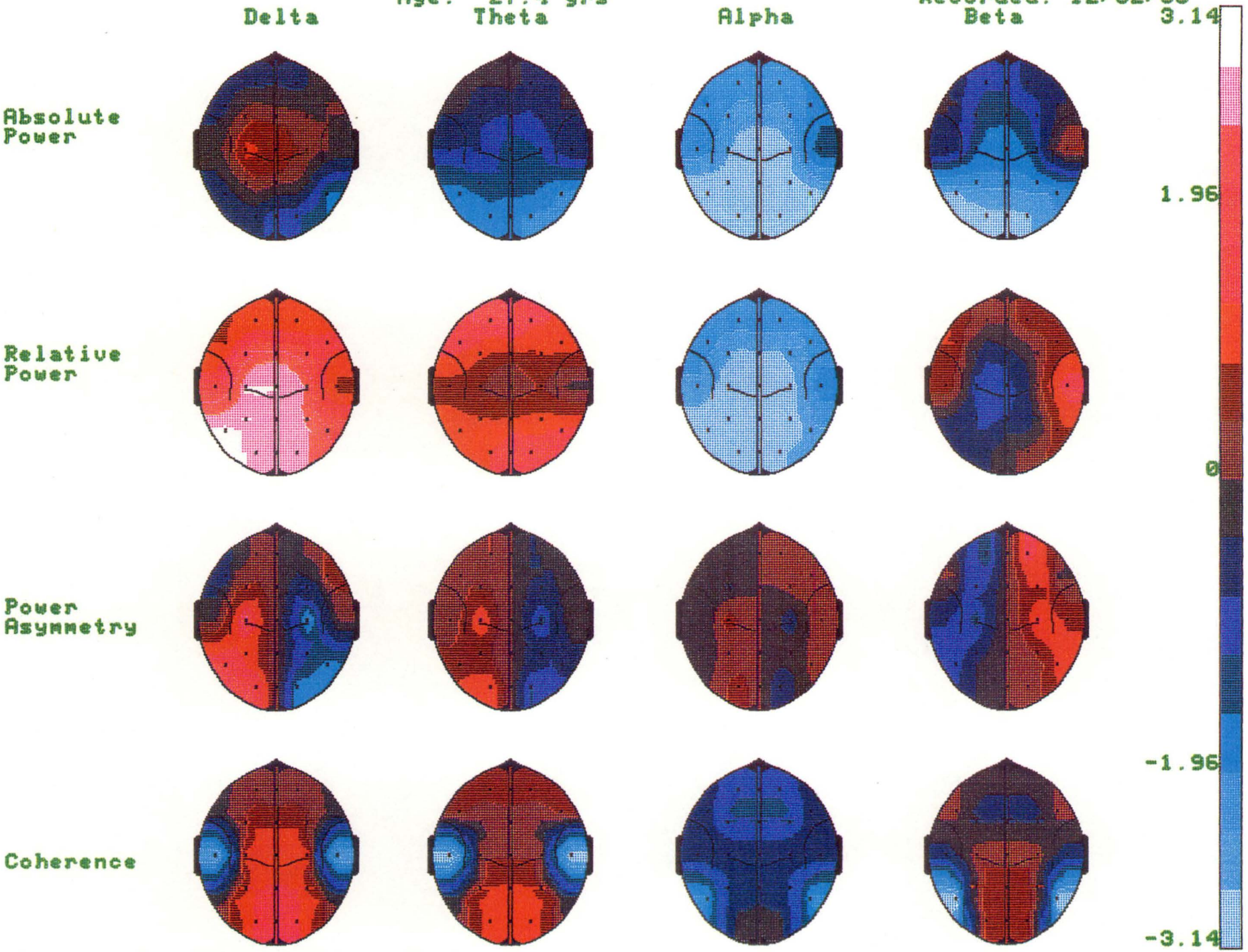
		T3	T4	T5	T6	P3	P4	O1	O2	Pz	Oz
bsolute	Δ	-0.18	-0.06	-0.60	-1.62	0.32	-0.06	-0.62	-1.14	0.39	-1.02
Power	θ	-0.80	-0.51	-1.77	-2.06	-1.10	-1.29	-1.83	-2.02	-1.09	-1.91
	α	-2.15	-1.25	-3.12	-2.89	-3.00	-3.02	-2.92	-3.16	-2.98	-3.15
	ß	-0.41	0.43	-2.80	-2.14	-2.27	-2.11	-2.87	-2.79	-2.10	-2.84
elative	Δ	1.48	0.66	2.93	1.95	2.89	2.64	2.76	2.57	2.67	2.61
Power	θ	0.60	-0.12	1.07	1.30	0.98	1.19	1.21	1.36	1.01	1.41
	α	-2.43	-1.69	-3.10	-2.42	-3.41	-3.28	-2.86	-2.84	-3.38	-2.95
	ß	0.73	1.51	-0.74	0.76	-0.91	-0.40	-0.47	0.11	-0.80	-0.10

Power	Δ	-0.03	1.65	1.05	1.82
ymmetry	θ	0.07	0.68	0.43	1.19
	α	-0.32	-0.27	0.11	0.73
	ß	-0.73	-1.04	-0.45	-0.07
herence	Δ	-2.75	-0.00	1.38	1.29
	θ	-3.55	-0.15	1.07	1.09
	α	-0.59	-2.12	-0.64	-0.18
	ß	-0.55	-3.12	0.80	0.55

Monopolar Z Score Maps

Name: GK F
Age: 27.4 yrs

Analyzed: 05/30/90
Recorded: 12/02/88



ne: GK F
e: 27.4 yrs

Analyzed: 05/30/90 Recorded: 12/02/88
Epochs: 38

polar Raw Measures

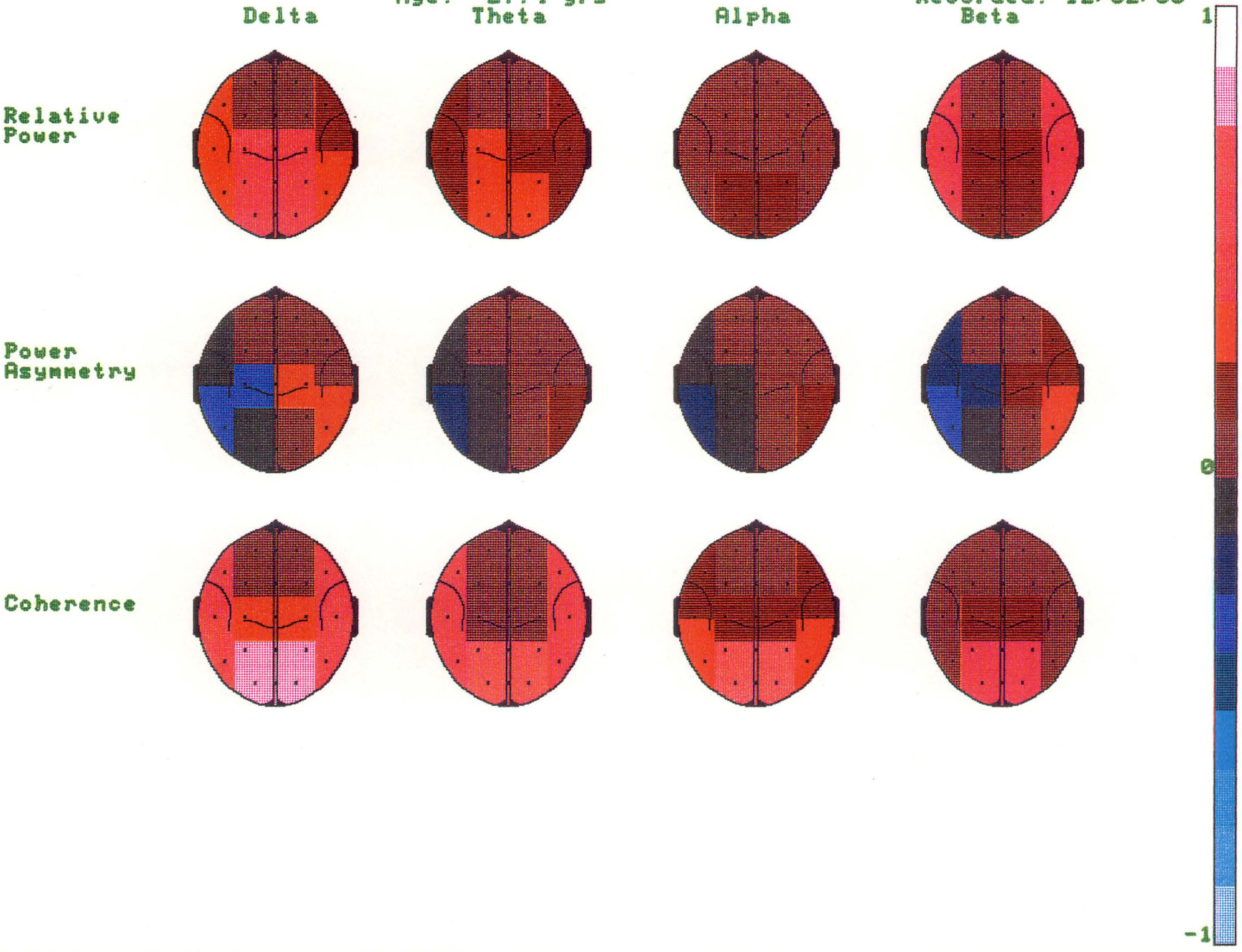
		Central		Temporal		Parietal Occipital		Frontal Temporal	
		C3/Cz	C4/Cz	T3/T5	T4/T6	P3/O1	P4/O2	F7/T3	F8/T
bsolute	Δ	1.7	3.3	2.4	4.8	2.3	2.5	2.0	2.
Power	θ	1.4	1.6	1.6	2.1	1.5	1.6	1.5	1.
(uU²)	α	0.6	0.6	0.8	1.3	0.8	0.9	0.9	1.
	β	0.9	1.3	3.0	5.1	0.8	0.9	3.0	4.
tal Absolute Power		4.5	6.8	7.8	13.3	5.4	5.9	7.5	9.
tal Power Asymmetry		-20.4		-26.3		-4.3		-12.6	
elative	Δ	37.9	49.1	30.6	36.1	41.7	42.0	27.1	23.
Power	θ	30.2	23.3	20.0	15.7	28.2	27.3	20.4	16.
(%)	α	12.4	8.7	10.8	9.9	14.5	15.1	12.1	11.
	β	19.5	18.9	38.5	38.2	15.5	15.6	40.4	48.
	Δ+θ	68.1	72.4	50.7	51.8	70.0	69.3	47.4	39.
	Combination	0.7	1.3	0.9	1.1	0.9	0.9	0.6	0.

Power	Δ	-32.5	-33.8	-4.6	-5.2
ymmetry	θ	-7.6	-14.8	-2.6	-1.3
(%)	α	-3.2	-22.2	-6.4	-11.7
	β	-19.0	-26.0	-4.5	-21.5
	Combination	1.1	0.8	-1.4	0.1
herence	Δ	28.3	53.8	78.1	39.0
(%)	θ	8.8	40.9	70.4	40.7
	α	13.3	26.8	65.2	22.8
	β	22.7	4.5	42.5	7.4
	Combination	0.8	1.2	0.6	0.8
erall		3.0	3.1	2.4	2.2

Bipolar Raw Maps

Name: GK F
Age: 27.4 yrs

Analyzed: 05/30/90
Recorded: 12/02/88



ne: GK F
e: 27.4 yrs

Analyzed: 05/30/90 Recorded: 12/02/88
Epochs: 38

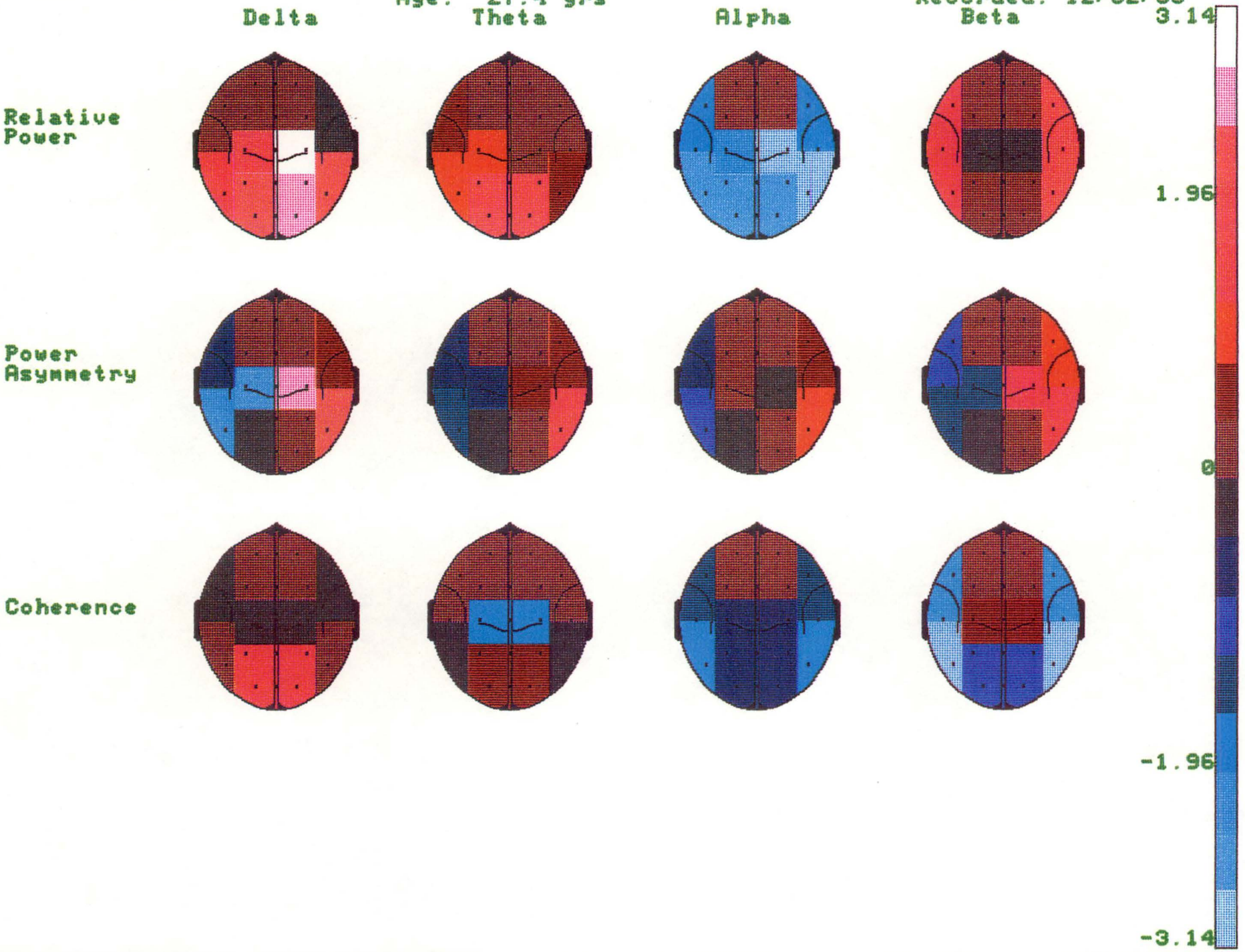
olar Normative Measures

		Central		Temporal		Parietal Occipital		Frontal Temporal	
		C3/Cz	C4/Cz	T3/T5	T4/T6	P3/O1	P4/O2	F7/T3	F8/T
al Absolute Power		15.1	15.8	26.3	26.3	23.4	25.1	14.8	14.1
al Power Asymmetry		-2.7		0.2		-3.3		2.2	
elative	Δ	19.2	18.2	14.5	13.2	11.7	11.6	24.6	24.4
ower	θ	22.4	21.8	14.0	12.3	13.2	13.0	17.5	16.2
(%)	α	34.1	35.1	48.9	53.0	56.5	57.0	29.6	31.5
	β	20.3	20.7	17.6	16.7	14.6	14.1	24.2	23.4
	Δ+θ	43.1	41.3	29.6	26.4	26.0	25.8	43.3	41.8
	Combination	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
ower	Δ	-0.5		2.6		-3.1		0.9	
mmetry	θ	-1.9		5.3		-1.9		5.1	
(%)	α	-3.6		-3.0		-4.1		0.6	
	β	-4.2		2.2		-2.1		4.2	
	Combination	0.4		0.4		0.4		0.4	
erence	Δ	31.6		53.0		62.3		43.2	
(%)	θ	29.5		44.2		66.1		38.7	
	α	19.3		56.6		74.2		40.3	
	β	15.0		25.9		54.1		19.4	
	Combination	0.5		0.5		0.4		0.4	
erall		2.1		2.1		2.1		2.0	

Bipolar Z Score Maps

Name: GK F
Age: 27.4 yrs

Analyzed: 05/30/90
Recorded: 12/02/88



ame: GK F
ge: 27.4 yrs

Analyzed: 05/30/90 Recorded: 12/02/88
Epochs: 38

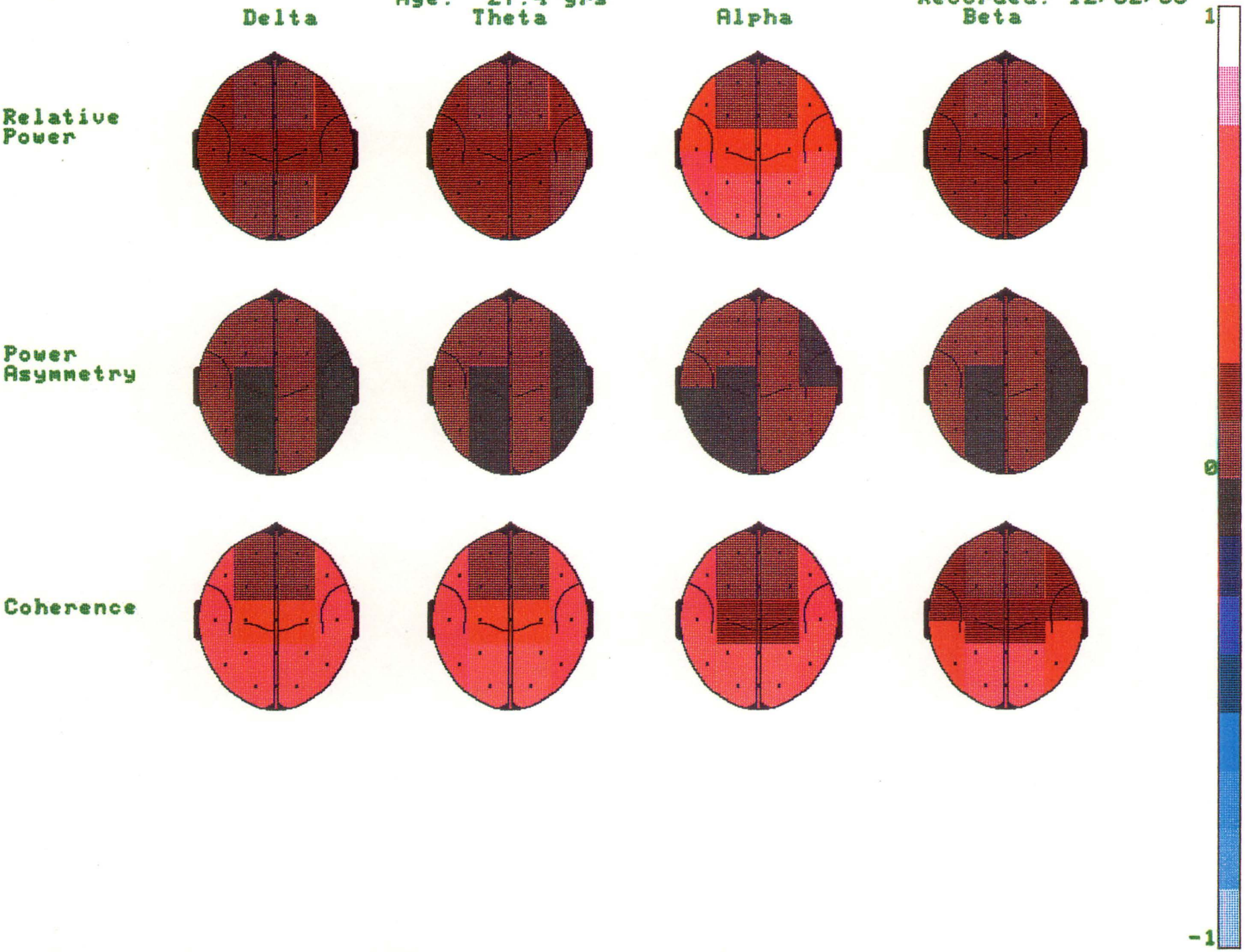
olar Z Score Measures

		Central		Temporal		Parietal Occipital		Frontal Temporal	
		C3/Cz	C4/Cz	T3/T5	T4/T6	P3/O1	P4/O2	F7/T3	F8/T4
al Absolute Power		-2.11	-1.47	-1.62	-0.86	-1.76	-1.78	-1.31	-0.72
al Power Asymmetry		-1.61		-1.58		-0.06		-1.11	
elative Power	Δ	1.72	2.83	1.48	2.07	2.35	2.48	0.26	-0.1
	θ	1.05	0.21	0.86	0.55	1.68	1.63	0.55	0.0
	α	-2.25	-2.95	-2.60	-2.94	-2.66	-2.65	-1.82	-1.9
	β	-0.11	-0.26	1.54	1.61	0.11	0.19	1.34	1.9
	Δ+θ	1.88	2.44	1.39	1.71	2.69	2.71	0.34	-0.1
	Combination	1.06	2.41	1.37	2.12	1.42	1.44	0.78	1.2
Power Asymmetry	Δ	-2.73		-2.15		-0.08		-0.43	
	θ	-0.58		-1.21		-0.05		-0.56	
	α	0.03		-0.92		-0.12		-0.69	
	β	-1.24		-1.38		-0.15		-1.07	
	Combination	1.42		0.80		-4.22		-0.75	
herence	Δ	-0.18		0.07		1.22		-0.22	
	θ	-1.88		-0.36		0.47		0.17	
	α	-0.55		-1.81		-0.67		-1.20	
	β	0.60		-3.62		-1.12		-2.03	
	Combination	0.96		1.92		0.38		1.00	
erall		2.39		2.77		0.74		0.52	

Bipolar Normative Maps

Name: GK F
Age: 27.4 yrs

Analyzed: 05/30/90
Recorded: 12/02/88



:e: GK F Analyzed: 05/30/90 Recorded: 12/02/88
: 27.4 yrs # Epochs: 38

olar Multivariate Z score Measures

		L HEM	R HEM	BACK	FRONT	ALL
al Absolute Power		1.46	0.92	1.44	0.81	1.31
al Power Asymmetry				0.69	0.99	0.92
elative	Δ	1.26	2.45	1.86	1.32	2.13
ower	θ	0.11	0.50	1.09	-0.96	0.40
	α	1.28	1.95	1.67	1.51	1.50
	ß	0.66	0.93	-2.51	0.38	0.31
	Δ+θ	1.58	1.88	2.09	0.64	1.91
	Combination	1.89	1.54	1.38	1.12	-1.00
ower	Δ			1.35	1.15	1.65
mmetry	θ			-0.88	0.36	-0.43
	α			-2.93	0.16	-0.81
	ß			0.29	0.89	0.60
	Combination			0.84	0.21	0.55
erence	Δ			0.30	-1.79	-0.55
	θ			0.97	-1.49	0.35
	α			-0.21	1.13	0.77
	ß			0.27	2.05	2.08
	Combination			0.73	1.72	1.76
erall				1.45	1.77	0.67

DEPARTMENT OF CLINICAL NEUROPHYSIOLOGY

CADWELL SPECTRUM 32

Patient Clinical Record

05/23/90

Neurometric Analysis (QEEG) Discriminants

Classification functions in this program were derived from groups of patients who met specific criteria for group membership.

Neurometric classifications are statistical in nature and serve only as an adjunct to the other clinical results used to evaluate the patient.

This patient's discriminant scores lie outside of the normal limits expected for an individual of this age.

ame: GK2 F Analyzed: 06/12/90 Recorded: 05/23/90
e: 28.9 yrs # Epochs: 38 Site Id: RHH

opolar Raw Measures

		Fp1	Fp2	F7	F8	F3	F4	C3	C4	Fpz	Fz	Cz
bsolute	Δ	14.3	12.6	15.0	9.2	27.0	20.7	30.4	23.9	15.6	29.3	35.3
ower	θ	5.5	5.3	5.6	3.8	8.9	7.4	9.7	7.9	5.5	9.2	11.0
uV²)	α	1.2	1.3	1.3	1.2	1.6	1.6	1.7	1.7	1.4	1.7	1.9
	β	1.5	2.8	2.7	1.9	2.4	2.7	2.0	2.4	2.0	2.0	2.2
	T	22.5	22.0	24.6	16.2	39.9	32.3	43.7	35.9	24.6	42.3	50.5
elative	Δ	63.4	57.3	60.7	57.0	67.6	64.0	69.4	66.6	63.5	69.3	70.0
ower	θ	24.5	24.0	22.9	23.5	22.3	22.8	22.1	21.9	22.6	21.8	21.8
(%)	α	5.2	6.1	5.3	7.5	4.1	4.9	3.9	4.8	5.6	4.1	3.9
	β	6.8	12.5	11.0	11.9	6.0	8.3	4.5	6.7	8.3	4.8	4.3

ower	Δ	6.3	23.6	13.2	11.9
mmetry	θ	2.2	19.4	9.3	10.4
(%)	α	-6.2	3.3	0.9	-0.3
	β	-28.6	16.7	-5.7	-9.7
erence	Δ	96.4	69.3	94.0	94.3
(%)	θ	93.5	57.3	88.4	86.9
	α	85.9	28.9	78.4	75.5
	β	63.8	20.9	62.8	70.9

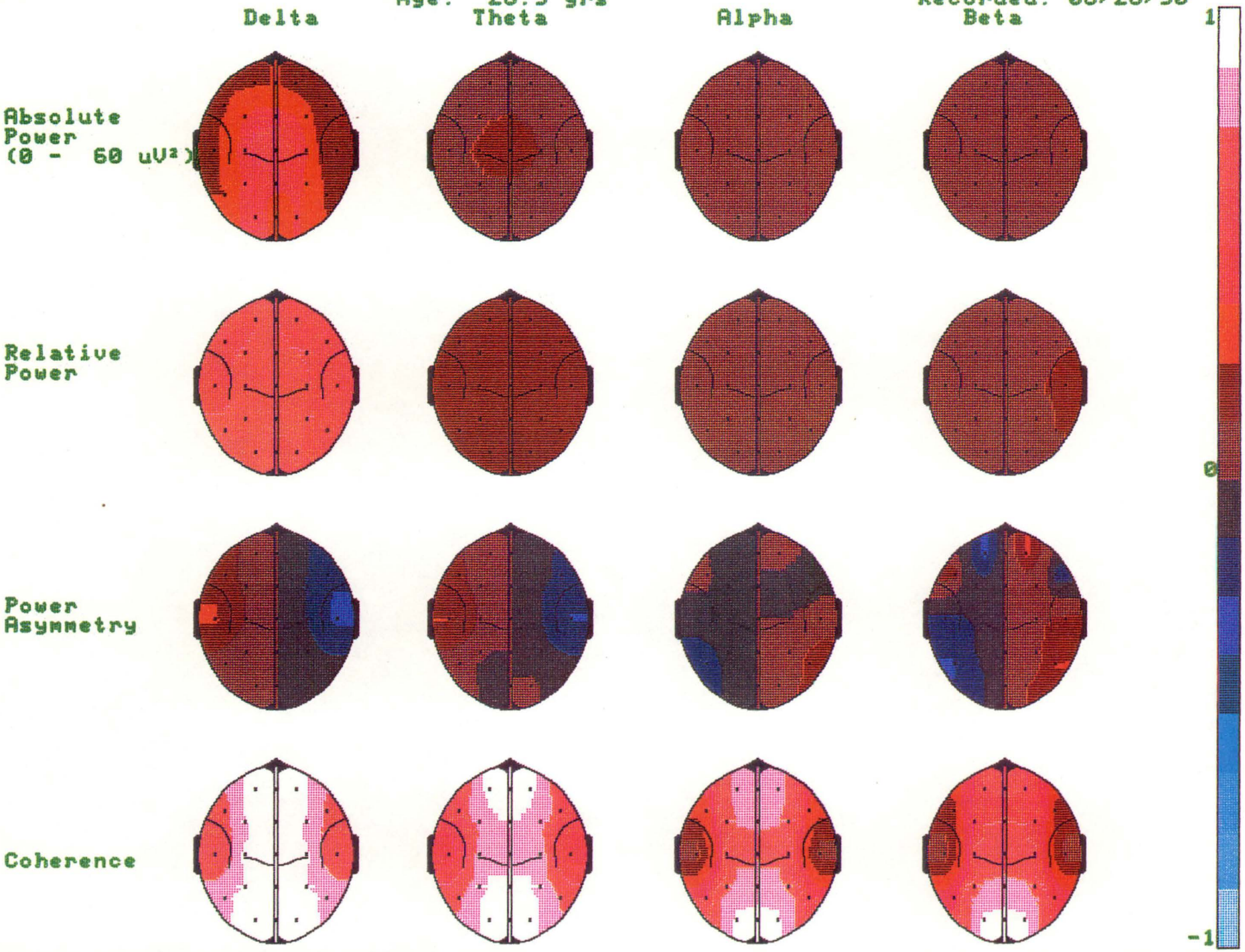
		T3	T4	T5	T6	P3	P4	O1	O2	Pz	Oz
bsolute	Δ	13.3	7.6	15.5	13.1	26.2	25.3	29.0	23.7	26.1	18.2
Power	θ	4.8	2.9	4.9	4.2	8.0	7.5	7.0	7.3	7.5	5.8
uV²)	α	1.4	1.6	1.5	2.3	2.0	2.2	2.4	2.7	2.3	2.8
	β	2.3	3.1	1.7	2.9	2.0	2.5	2.2	2.9	2.5	3.0
	T	21.9	15.2	23.6	22.4	38.2	37.5	40.6	36.6	38.3	29.7
elative	Δ	60.7	49.8	65.8	58.3	68.7	67.4	71.4	64.7	68.0	61.2
Power	θ	22.1	19.0	20.6	18.5	21.0	20.1	17.2	20.0	19.5	19.4
(%)	α	6.6	10.6	6.4	10.3	5.2	5.9	5.9	7.4	5.9	9.4
	β	10.6	20.6	7.2	12.9	5.1	6.6	5.5	7.9	6.5	10.0

Power	Δ	27.5	8.6	1.8	10.2
mmetry	θ	25.3	8.0	3.1	-2.4
(%)	α	-5.1	-20.9	-6.0	-6.2
	β	-15.0	-26.0	-11.6	-12.6
herence	Δ	63.1	85.4	96.5	93.5
(%)	θ	42.9	73.6	92.2	93.5
	α	5.9	51.0	83.2	92.4
	β	5.0	46.7	81.4	92.3

Monopolar Raw Maps

Name: GK2 F
Age: 28.9 yrs

Analyzed: 06/12/90
Recorded: 05/23/90



ne: GK2 F
e: 28.9 yrs

Analyzed: 06/12/90
Epochs: 38

Recorded: 05/23/90
Site Id: RHH

nopolar Normative Measures

		Fp1	Fp2	F7	F8	F3	F4	C3	C4	Fpz	Fz	Cz
solute	Δ	9.9	9.6	6.4	6.0	10.6	10.2	10.3	10.6	9.7	12.6	15.5
ower	θ	6.7	6.6	5.0	4.7	9.7	9.3	9.7	9.9	6.6	12.4	15.3
uU²)	α	8.9	8.7	6.7	6.6	12.5	12.2	14.7	15.6	8.8	15.3	20.7
	ß	4.6	4.5	3.7	3.5	6.1	6.0	6.5	6.6	4.5	6.6	8.4
lative	Δ	31.8	31.7	28.2	28.0	25.8	25.7	23.8	23.8	31.7	25.6	24.7
ower	θ	21.1	21.1	21.7	21.5	23.7	23.4	22.2	22.0	21.1	25.2	24.1
(%)	α	28.7	28.9	30.2	31.1	31.6	31.8	34.8	35.5	28.8	31.9	33.5
	ß	14.6	14.4	16.3	16.3	15.0	15.4	14.9	14.6	14.5	13.2	13.4

ower	Δ	1.6	2.9	2.0	-1.8
mmetry	θ	1.4	3.1	2.3	-1.5
(%)	α	0.9	1.1	1.3	-2.9
	ß	1.7	2.5	0.4	-0.9
erence	Δ	87.4	28.8	83.5	82.2
(%)	θ	86.4	29.7	83.0	76.6
	α	92.7	52.4	88.2	68.1
	ß	78.5	29.1	73.2	54.4

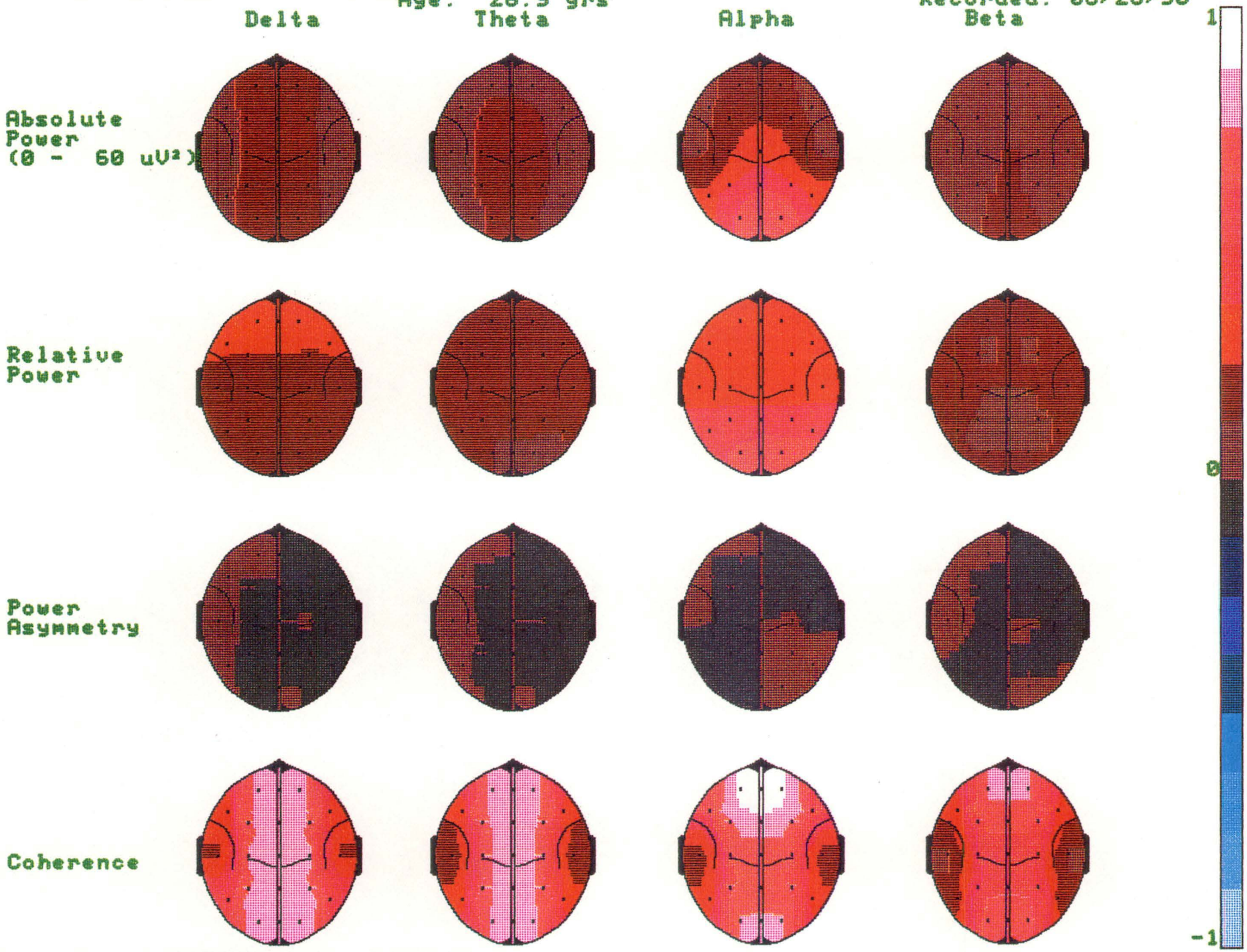
		T3	T4	T5	T6	P3	P4	O1	O2	Pz	Oz
solute	Δ	4.7	4.2	7.1	6.8	11.2	11.0	9.3	9.4	13.7	9.4
ower	θ	4.1	3.7	6.4	6.0	9.8	9.7	8.2	8.1	12.4	8.2
uU²)	α	6.5	6.2	17.1	20.3	25.5	26.8	32.8	37.3	31.3	34.9
	ß	4.1	3.6	6.2	6.2	8.2	8.3	8.2	8.6	9.0	8.4
lative	Δ	23.1	22.0	18.2	16.4	19.1	18.4	15.0	13.8	19.2	14.4
ower	θ	20.2	18.8	16.2	14.1	16.7	16.0	13.0	12.1	17.2	12.5
(%)	α	32.4	32.8	45.5	50.3	45.4	47.0	54.6	57.1	46.2	55.9
	ß	20.4	18.9	15.7	14.9	13.9	13.6	13.2	12.7	12.6	13.0

ower	Δ	4.9	1.8	0.4	-0.3
mmetry	θ	5.8	3.8	0.8	-0.2
(%)	α	2.3	-7.9	-3.3	-6.4
	ß	6.7	-0.4	-0.7	-2.3
herence	Δ	25.0	55.4	85.7	86.8
(%)	θ	13.7	32.9	78.5	81.2
	α	18.1	29.8	70.4	77.6
	ß	10.4	17.6	61.4	70.3

Monopolar Normative Maps

Name: GK2 F
Age: 28.9 yrs

Analyzed: 06/12/90
Recorded: 05/23/90



ɯe: GK2 F
: 28.9 yrs

Analyzed: 06/12/90
Epochs: 38

Recorded: 05/23/90
Site Id: RHH

ıopolar Z Score Measures

		Fp1	Fp2	F7	F8	F3	F4	C3	C4	Fpz	Fz	Cz
·solute	Δ	0.69	0.54	1.96	0.93	2.39	1.73	2.63	1.93	0.91	2.05	1.98
'ower	θ	-0.41	-0.45	0.29	-0.41	-0.19	-0.52	-0.01	-0.48	-0.37	-0.65	-0.73
	α	-2.63	-2.43	-2.18	-2.14	-2.64	-2.63	-2.72	-2.77	-2.40	-2.78	-2.97
	β	-2.30	-1.04	-0.61	-1.16	-1.78	-1.54	-2.09	-1.84	-1.70	-2.14	-2.37
elative	Δ	2.43	2.01	2.91	2.58	3.60	3.27	3.74	3.50	2.47	3.68	3.61
'ower	θ	0.60	0.52	0.22	0.40	-0.22	-0.10	-0.01	-0.01	0.27	-0.52	-0.35
	α	-3.13	-2.88	-3.51	-2.99	-3.83	-3.58	-4.20	-3.95	-3.01	-3.85	-3.92
	β	-1.87	-0.38	-1.04	-0.87	-2.29	-1.62	-2.79	-1.91	-1.43	-2.50	-2.65

		Fp1-Fp2	F7-F8	F3-F4	C3-C4
ower	Δ	0.63	1.26	1.49	1.84
ɯmetry	θ	0.12	1.02	0.97	1.57
	α	-1.29	0.15	-0.05	0.28
	β	-3.82	0.89	-0.77	-0.97
ɯerence	Δ	2.16	1.62	2.49	2.97
	θ	1.63	1.16	1.09	1.94
	α	-1.12	-1.04	-1.23	0.56
	β	-1.20	-0.68	-0.95	1.40

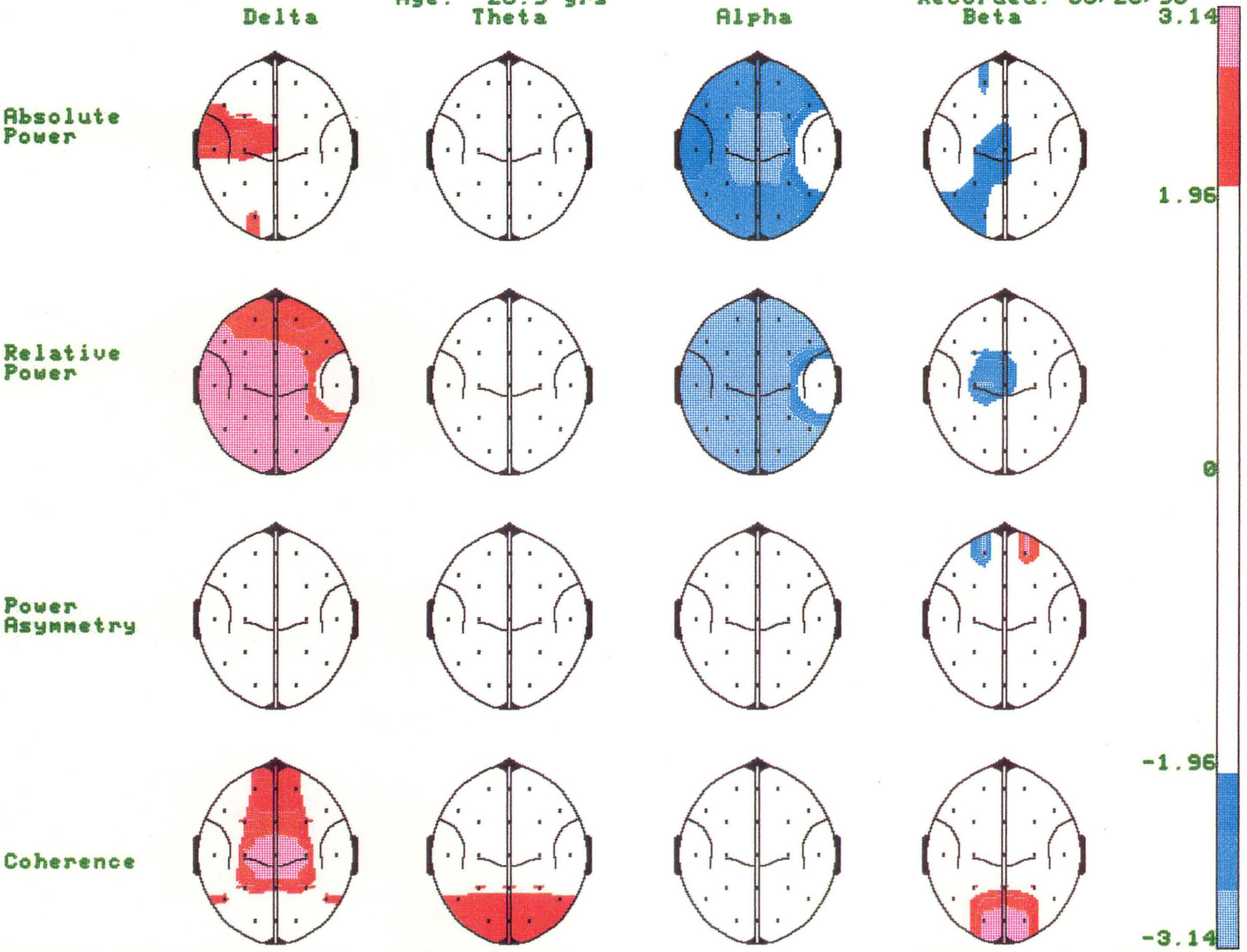
		T3	T4	T5	T6	P3	P4	O1	O2	Pz	Oz
ɔsolute	Δ	2.37	0.69	1.68	1.25	1.86	1.85	2.40	1.75	1.47	1.33
²ower	θ	0.33	-0.28	-0.48	-0.60	-0.39	-0.50	-0.28	-0.17	-0.99	-0.60
	α	-1.98	-1.24	-2.48	-2.13	-2.64	-2.57	-2.42	-2.46	-2.65	-2.35
	β	-0.97	-0.16	-2.07	-1.16	-2.28	-1.93	-2.02	-1.67	-2.04	-1.62
elative	Δ	3.54	1.88	3.66	3.08	3.54	3.44	3.75	3.36	3.23	3.13
²ower	θ	0.33	0.02	0.62	0.67	0.54	0.54	0.56	1.01	0.31	0.88
	α	-3.40	-1.87	-3.51	-2.97	-3.69	-3.60	-3.71	-3.56	-3.37	-3.15
	β	-1.44	0.16	-1.63	-0.30	-2.05	-1.58	-1.64	-0.92	-1.42	-0.51

		T3-T4	T5-T6	P3-P4	O1-O2
²ower	Δ	0.59	0.39	0.16	1.31
ɯmetry	θ	0.52	0.23	0.27	-0.26
	α	-0.18	-0.65	-0.21	0.02
	β	-0.50	-1.63	-1.01	-0.86
herence	Δ	1.75	2.09	2.65	1.25
	θ	1.62	2.11	2.32	2.30
	α	-1.58	0.93	1.09	1.83
	β	-1.12	1.77	2.11	3.36

Monopolar Z Score Maps

Name: GK2 F
Age: 28.9 yrs

Analyzed: 06/12/90
Recorded: 05/23/90



ne: GK2 F
e: 28.9 yrs

Analyzed: 06/12/90
Epochs: 38

Recorded: 05/23/90
Site Id: RHH

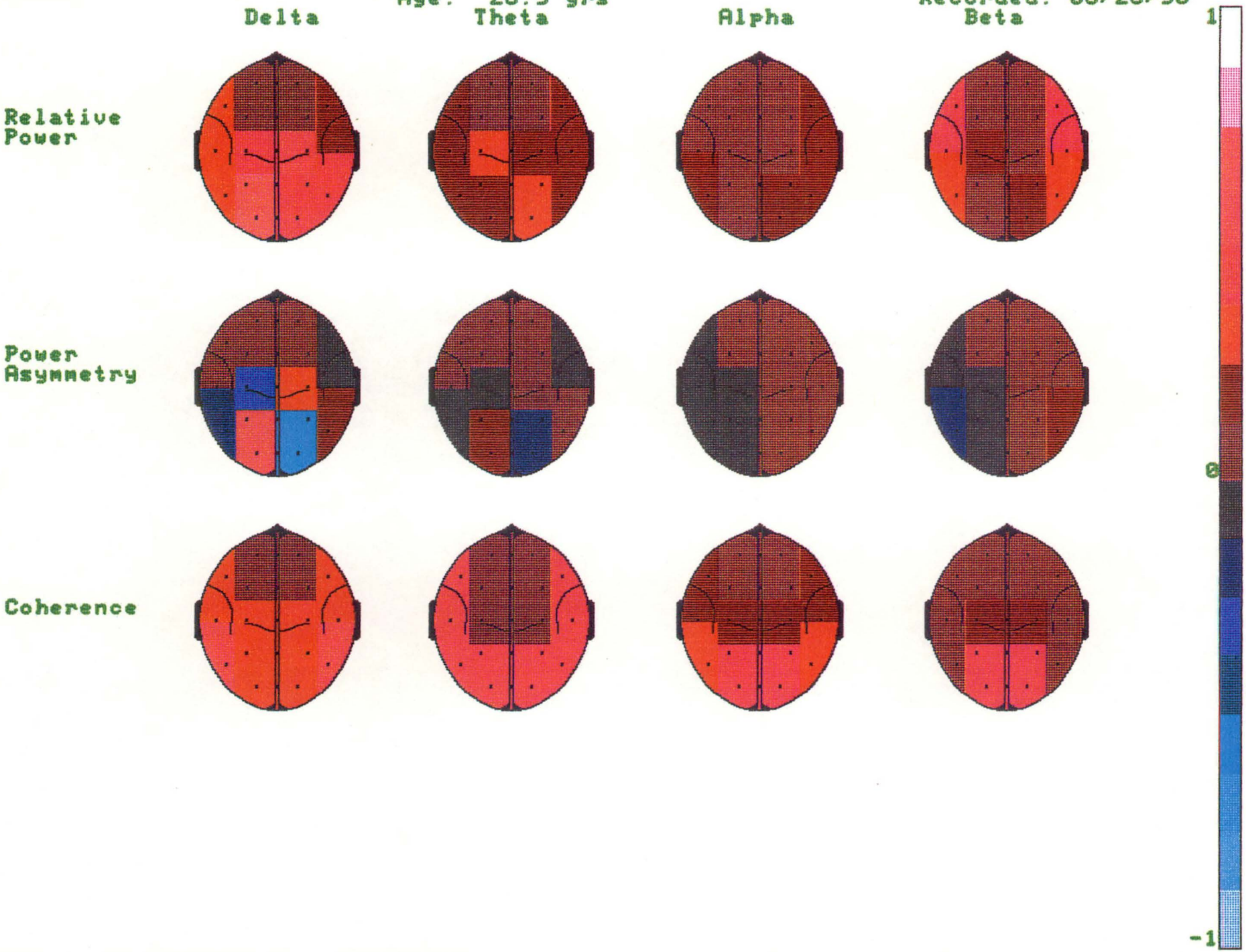
olar Raw Measures

		Central		Temporal		Parietal Occipital		Frontal Temporal	
		C3/Cz	C4/Cz	T3/T5	T4/T6	P3/O1	P4/O2	F7/T3	F8/T
solute	Δ	1.9	3.3	2.5	3.9	5.3	1.4	1.9	1.
ower	θ	1.1	1.3	1.4	1.5	1.3	0.9	1.5	1.
(uU²)	α	0.4	0.4	1.1	1.4	0.4	0.5	0.9	1.
	ß	0.5	0.6	2.0	2.8	0.5	0.6	3.0	3.
tal Absolute Power		3.9	5.6	7.0	9.5	7.5	3.3	7.2	7.
tal Power Asymmetry		-17.5		-15.1		38.9		-0.7	
elative	Δ	47.8	58.8	35.0	40.9	70.6	41.2	26.0	25.
ower	θ	28.9	23.0	20.5	15.4	16.7	25.9	20.4	17.
(%)	α	9.6	7.4	15.7	14.5	5.9	14.0	11.7	14.
	ß	13.6	10.8	28.8	29.1	6.7	18.8	41.8	43.
	Δ+θ	76.7	81.8	55.5	56.3	87.3	67.1	46.4	42.
	Combination	1.0	1.4	0.7	1.1	1.6	0.9	0.7	0.
ower	Δ	-27.4		-22.6		59.1		1.4	
mmetry	θ	-6.1		-1.0		18.9		6.8	
(%)	α	-4.2		-11.4		-2.1		-10.7	
	ß	-6.0		-15.6		-10.5		-2.3	
	Combination	0.8		0.6		1.3		-0.1	
herence	Δ	34.5		54.2		31.5		32.0	
(%)	θ	2.9		40.1		45.8		43.9	
	α	12.6		34.2		40.1		17.4	
	ß	24.7		12.5		41.4		5.7	
	Combination	1.4		0.5		1.0		1.1	
erall		3.6		2.4		3.6		2.6	

Bipolar Raw Maps

Name: GKZ F
Age: 28.9 yrs

Analyzed: 06/12/90
Recorded: 05/23/90



me: GK2 F Analyzed: 06/12/90 Recorded: 05/23/90
: 28.9 yrs # Epochs: 38 Site Id: RHH

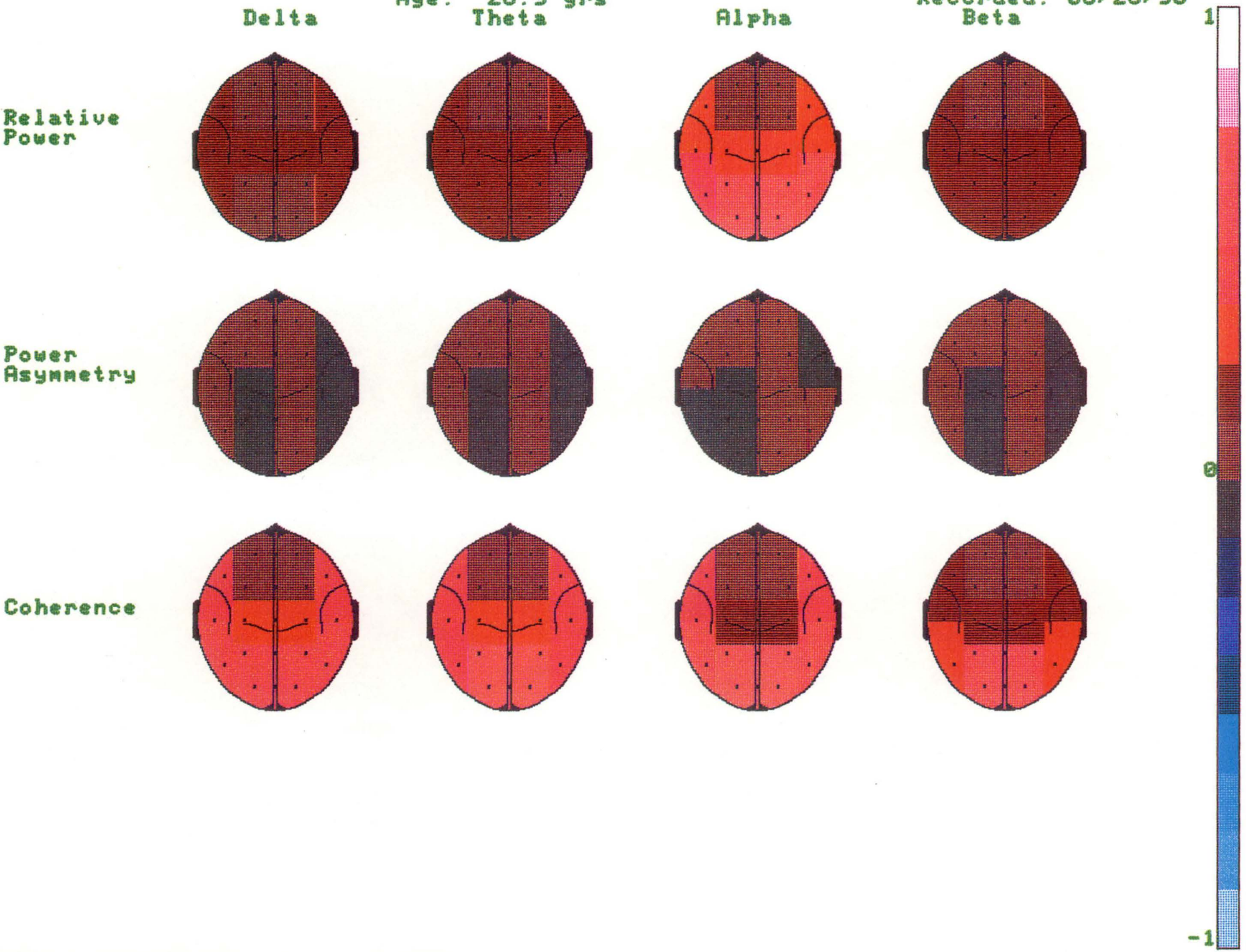
olar Normative Measures

		Central		Temporal		Parietal Occipital		Frontal Temporal	
		C3/Cz	C4/Cz	T3/T5	T4/T6	P3/01	P4/02	F7/T3	F8/T
al Absolute Power		15.1	15.8	26.3	26.3	23.4	25.1	14.8	14.
al Power Asymmetry		-2.7		0.2		-3.3		2.2	
lative	Δ	18.8	17.9	14.2	13.0	11.6	11.5	23.9	23.
ower	θ	21.9	21.3	13.8	12.2	13.2	12.9	17.3	16.
(%)	α	34.0	34.9	48.8	52.7	56.0	56.6	29.8	31.
	ß	21.0	21.5	18.0	17.1	15.1	14.5	24.7	23.
	Δ+θ	42.3	40.5	29.2	26.1	25.8	25.6	42.4	41.
	Combination	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.
ower	Δ	-0.5		2.6		-3.1		0.9	
mmetry	θ	-1.9		5.3		-1.9		5.1	
(%)	α	-3.6		-3.0		-4.1		0.6	
	ß	-4.2		2.2		-2.1		4.2	
	Combination	0.4		0.4		0.4		0.4	
erence	Δ	30.6		52.6		62.2		42.6	
(%)	θ	28.4		44.1		66.0		38.4	
	α	19.3		56.3		73.9		40.1	
	ß	14.8		25.9		53.7		19.4	
	Combination	0.5		0.5		0.4		0.4	
erall		2.1		2.1		2.1		2.0	

Bipolar Normative Maps

Name: GKZ F
Age: 28.9 yrs

Analyzed: 06/12/90
Recorded: 05/23/90



ıe: GK2 F
: 28.9 yrs

Analyzed: 06/12/90
Epochs: 38

Recorded: 05/23/90
Site Id: RHH

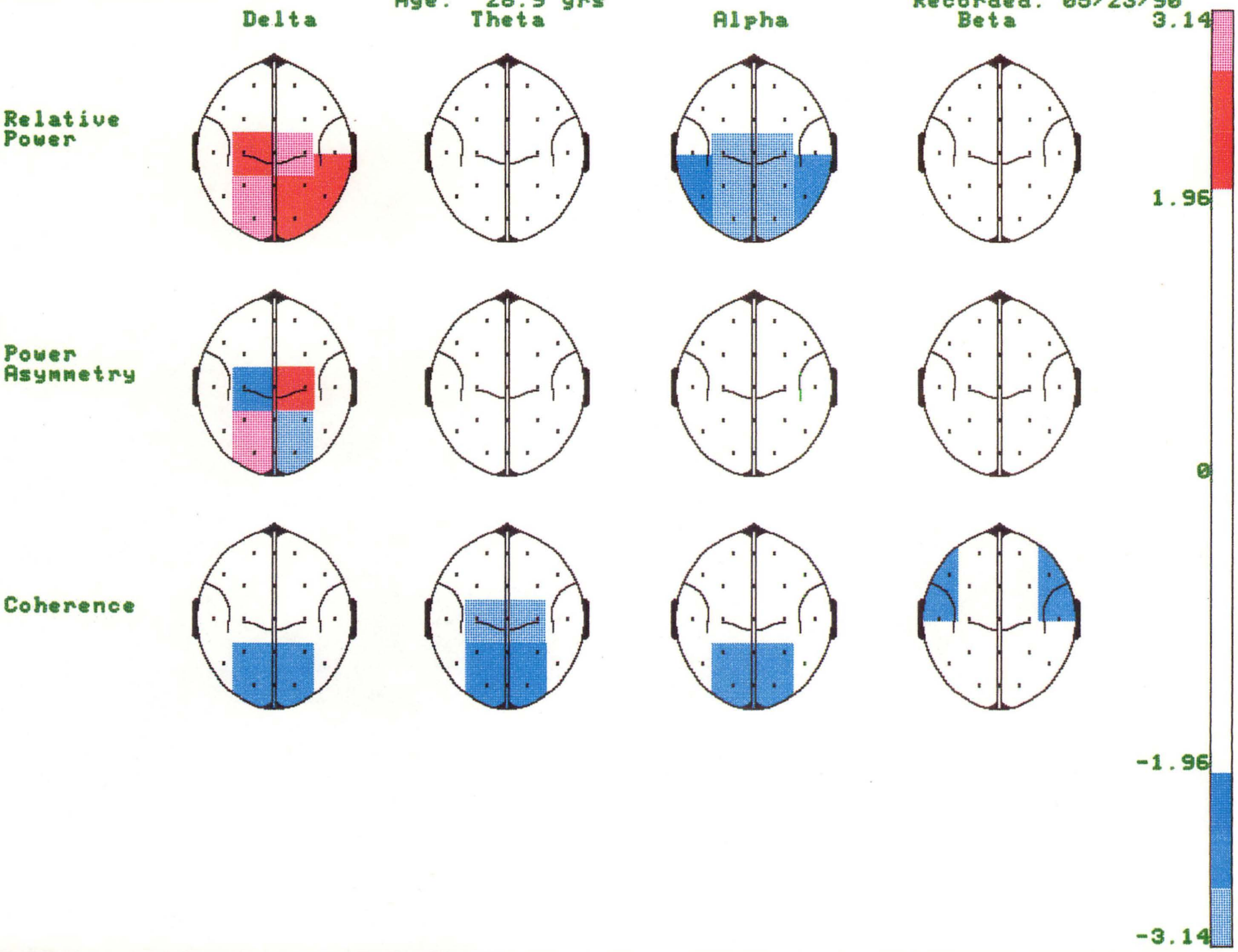
olar Z Score Measures

		Central		Temporal		Parietal Occipital		Frontal Temporal	
		C3/Cz	C4/Cz	T3/T5	T4/T6	P3/O1	P4/O2	F7/T3	F8/T4
al Absolute Power		-2.36	-1.82	-1.76	-1.29	-1.37	-2.50	-1.38	-1.25
al Power Asymmetry		-1.33		-0.90		2.78		-0.22	
lative ower	Δ	2.51	3.63	1.82	2.42	4.05	2.45	0.22	0.16
	θ	0.96	0.23	0.94	0.51	0.50	1.52	0.62	0.31
	α	-2.73	-3.25	-2.06	-2.38	-3.94	-2.75	-1.89	-1.57
	β	-1.11	-1.79	0.87	0.98	-1.47	0.49	1.39	1.53
	Δ+θ	2.72	3.50	1.73	2.01	4.24	2.58	0.33	0.16
	Combination	1.72	2.80	0.94	2.00	3.20	1.54	0.89	0.72
ower mmetry	Δ	-2.26		-1.45		3.71		0.03	
	θ	-0.43		-0.38		1.38		0.15	
	α	-0.04		-0.39		0.11		-0.63	
	β	-0.15		-0.86		-0.53		-0.26	
	Combination	0.95		0.45		2.12		-1.39	
ıerence	Δ	0.19		0.13		-2.36		-0.59	
	θ	-3.51		-0.42		-2.17		0.44	
	α	-0.62		-1.25		-2.42		-1.73	
	β	0.74		-1.52		-1.20		-2.58	
	Combination	2.49		0.09		1.55		1.83	
erall		4.10		0.91		3.53		1.38	

Bipolar Z Score Maps

Name: GKZ F
Age: 28.9 yrs

Analyzed: 06/12/90
Recorded: 05/23/90



e: GK2 F Analyzed: 06/12/90 Recorded: 05/23/90
: 28.9 yrs # Epochs: 38 Site Id: RHH

olar Multivariate Z score Measures

		L HEM	R HEM	BACK	FRONT	ALL
al Absolute Power		1.50	1.52	1.66	1.15	1.62
al Power Asymmetry				1.72	-0.21	1.48
lative ower	Δ	2.54	2.89	2.79	1.56	3.16
	θ	-0.92	0.26	1.82	-1.10	1.16
	α	2.48	2.08	2.63	1.19	3.07
	β	1.87	1.81	2.96	0.03	3.35
	Δ+θ	2.38	2.25	2.65	0.97	2.58
	Combination	-1.02	-0.29	0.43	-0.07	0.50
ower mmetry	Δ			1.94	0.55	2.26
	θ			0.51	-1.67	-0.34
	α			-3.05	-0.52	-1.76
	β			-1.05	-0.00	-0.82
	Combination			2.09	-0.36	1.76
nerence	Δ			1.42	-0.51	0.89
	θ			2.17	-0.81	2.11
	α			1.36	1.11	1.38
	β			0.44	1.58	1.48
	Combination			2.73	1.34	3.15
erall				1.87	0.80	1.73

DEPARTMENT OF NEUROPHYSIOLOGY

CADWELL SPECTRUM 32

Neurometric (QEEG) Discriminants

Stored T-Score data analysis

Name 1: GK1 F
a is GK1 compared to GK2 Name 2: K Gina
opolar Independent Tscore/Diff. Zscore Measures Analyzed: 06/12/90
rees of freedom: 74

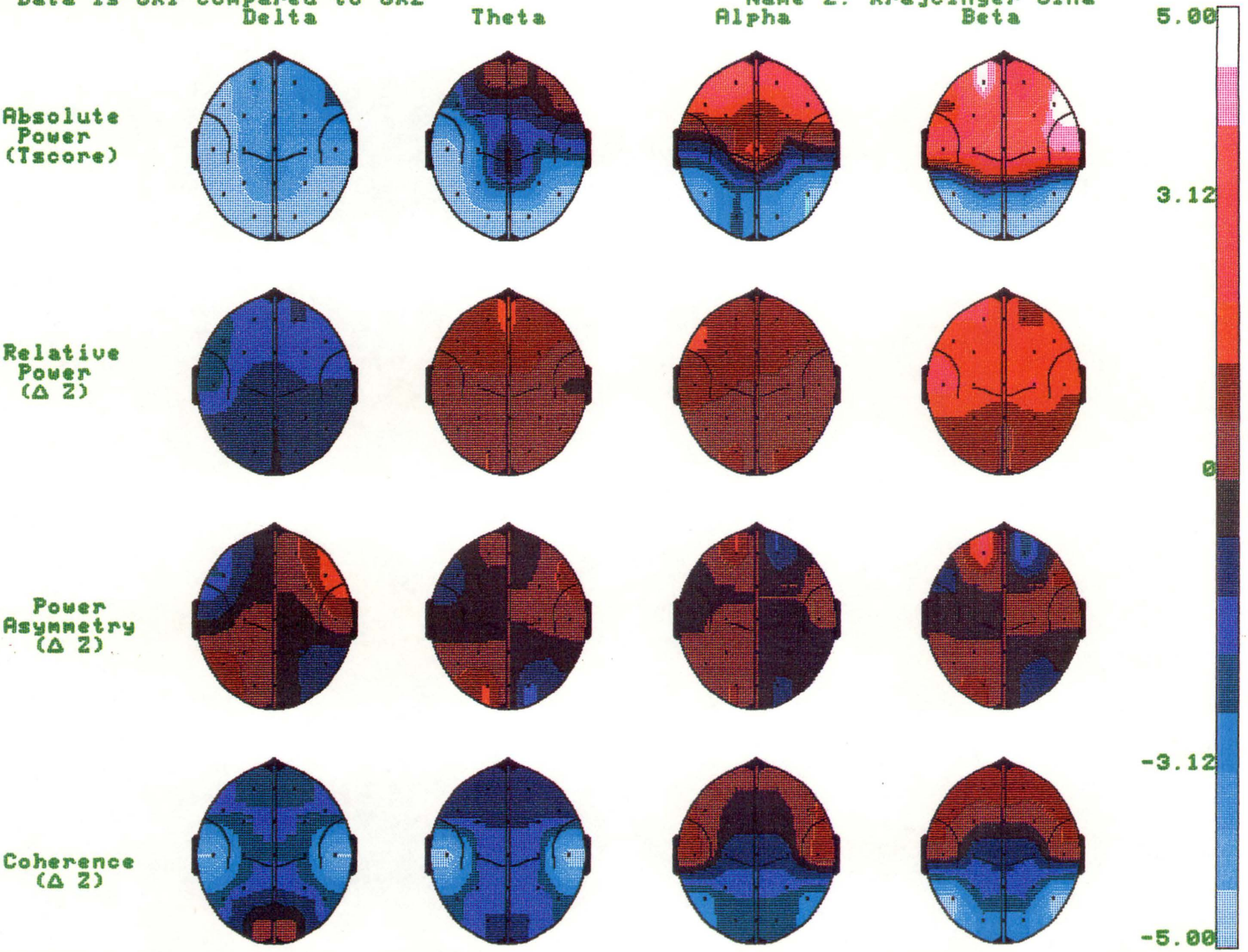
		Fp1	Fp2	F7	F8	F3	F4	C3	C4	Fpz	Fz	Cz
solute	Δ	-3.6	-2.9	-5.9	-2.4	-4.1	-3.3	-3.9	-3.9	-4.2	-3.4	-3.7
ower	θ	0.2	-0.0	-2.2	0.3	-0.9	-0.3	-2.9	-2.5	0.4	-2.3	-0.4
score)	α	3.7	2.2	1.8	2.2	1.7	2.5	-0.2	-1.0	2.1	-0.4	1.9
	ß	5.7	1.9	1.1	5.0	2.2	3.5	2.8	3.6	2.6	3.1	3.7
lative	Δ	-1.5	-1.2	-2.1	-1.3	-1.4	-1.5	-0.9	-1.2	-1.5	-1.4	-0.8
ower	θ	1.1	0.9	1.3	0.6	1.1	1.0	0.5	0.5	1.5	1.1	0.5
Δ Z)	α	1.0	0.7	1.4	0.7	1.0	1.0	0.8	0.6	0.9	0.9	0.6
	ß	2.0	1.2	1.8	1.4	1.6	1.6	1.8	2.1	1.5	2.0	1.7

ower	Δ	-0.5	-1.9	-0.4	0.4
mmetry	θ	0.3	-0.9	-0.5	-0.1
Δ Z)	α	1.3	-0.2	-0.5	0.5
	ß	2.3	-0.8	-0.4	-0.4
erence	Δ	-2.0	-1.7	-1.6	-1.9
Δ Z)	θ	-1.0	-0.6	-1.1	-1.6
	α	0.0	0.6	-0.1	-1.0
	ß	0.9	0.8	0.4	-1.1

		T3	T4	T5	T6	P3	P4	O1	O2	Pz	Oz
solute	Δ	-6.2	-4.0	-6.4	-8.1	-4.4	-5.0	-8.0	-7.5	-2.9	-6.7
ower	θ	-5.0	-1.7	-6.2	-6.3	-3.1	-3.2	-6.6	-7.8	-0.3	-6.5
score)	α	-1.2	-0.1	-4.3	-4.9	-2.6	-3.0	-2.2	-3.3	-2.2	-3.4
	ß	2.9	3.9	-6.9	-8.0	-0.2	-1.6	-7.0	-7.7	-0.6	-8.8
lative	Δ	-2.1	-1.2	-0.7	-1.1	-0.6	-0.8	-1.0	-0.8	-0.6	-0.5
ower	θ	0.3	-0.1	0.5	0.6	0.4	0.6	0.7	0.3	0.7	0.5
Δ Z)	α	1.0	0.2	0.4	0.6	0.3	0.3	0.9	0.7	-0.0	0.2
	ß	2.2	1.4	0.9	1.1	1.1	1.2	1.2	1.0	0.6	0.4

ower	Δ	-0.6	1.3	0.9	0.5
mmetry	θ	-0.5	0.5	0.2	1.5
Δ Z)	α	-0.1	0.4	0.3	0.7
	ß	-0.2	0.6	0.6	0.8
herence	Δ	-4.5	-2.1	-1.3	0.0
Δ Z)	θ	-5.2	-2.3	-1.2	-1.2
	α	1.0	-3.1	-1.7	-2.0
	ß	0.6	-4.9	-1.3	-2.8

Monopolar Independent Tscore/Diff. Zscore Maps Name 1: GK1 F
Data is GK1 compared to GK2 Name 2: Krajcinger Gina



Name 1: GK1 F
:a is GK1 compared to GK2 Name 2: K
olar Independent Tscore/Diff. Zscore Measures Analyzed: 06/12/90
rees of freedom: 74

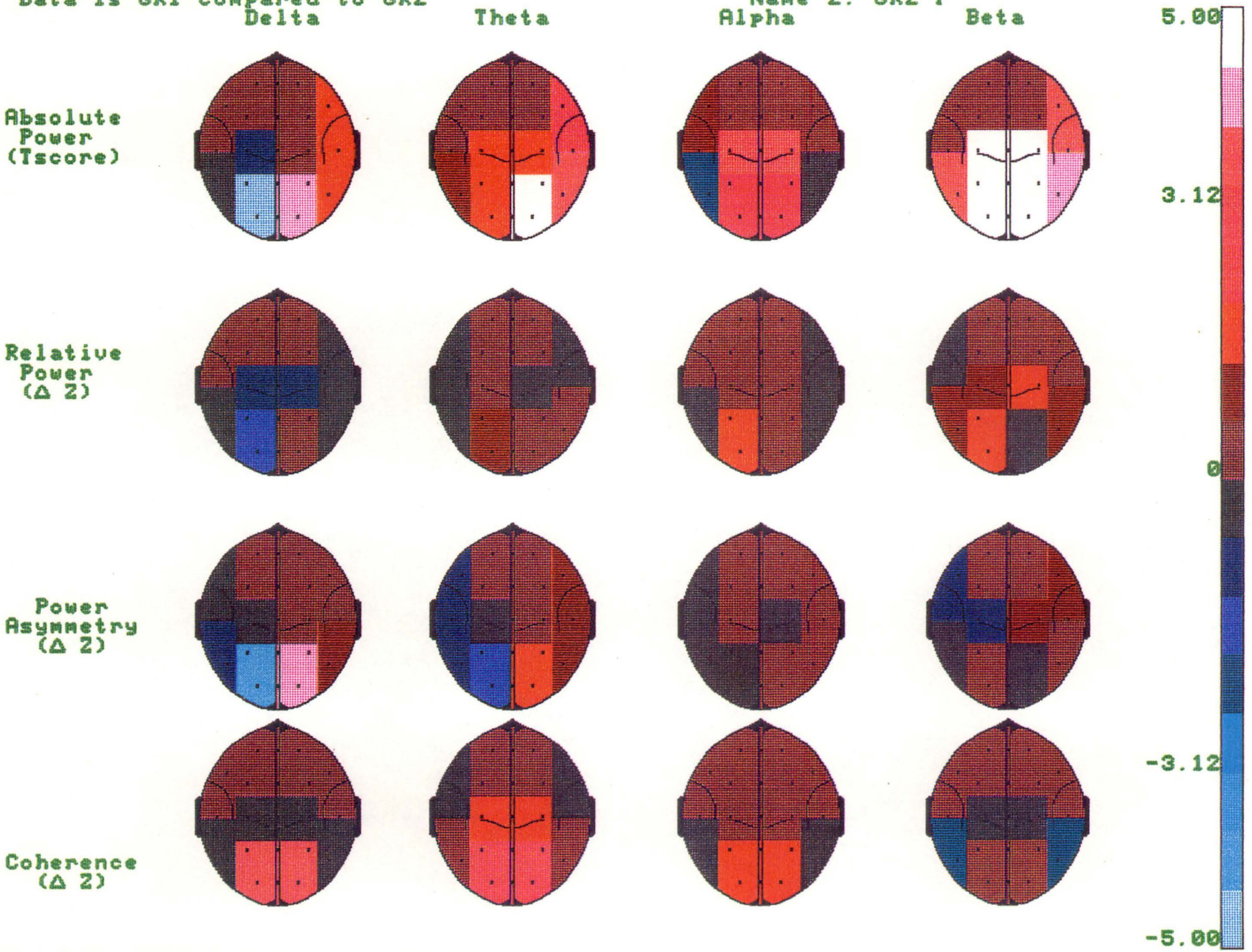
		Central		Temporal		Parietal Occipital		Frontal Temporal	
		C3/Cz	C4/Cz	T3/T5	T4/T6	P3/O1	P4/O2	F7/T3	F8/T4
solute	Δ	-0.7	0.2	-0.2	1.5	-6.1	4.0	0.6	1.3
ower	θ	1.7	1.8	0.9	3.3	1.8	5.3	0.4	2.0
score)	α	3.0	2.6	-2.4	-0.3	1.9	2.5	0.7	0.4
	β	6.3	6.5	3.5	4.2	6.4	5.3	0.1	3.3
elative	Δ	-0.8	-0.8	-0.3	-0.3	-1.7	0.0	0.0	-0.2
ower	θ	0.1	-0.0	-0.1	0.0	1.2	0.1	-0.1	-0.3
(Δ Z)	α	0.5	0.3	-0.5	-0.6	1.3	0.1	0.1	-0.3
	β	1.0	1.5	0.7	0.6	1.6	-0.3	-0.0	0.4

Power	Δ	-0.5	-0.7	-3.8	-0.5
ymmetry	θ	-0.1	-0.8	-1.4	-0.7
(Δ Z)	α	0.1	-0.5	-0.2	-0.1
	β	-1.1	-0.5	0.4	-0.8

herence	Δ	-0.4	-0.1	3.6	0.4
(Δ Z)	θ	1.6	0.1	2.6	-0.3
	α	0.1	-0.6	1.8	0.5
	β	-0.1	-2.1	0.1	0.5

Bipolar Independent Tscore/Diff. Zscore Maps
Data is GK1 compared to GK2

Name 1: GK1 F
Name 2: GK2 F



Name 1: GK1 F
a is GK1 compared to GK2 Name 2:
opolar Correlated Tscore/Diff. Zscore Measures Analyzed: 06/12/90
rees of freedom: 37

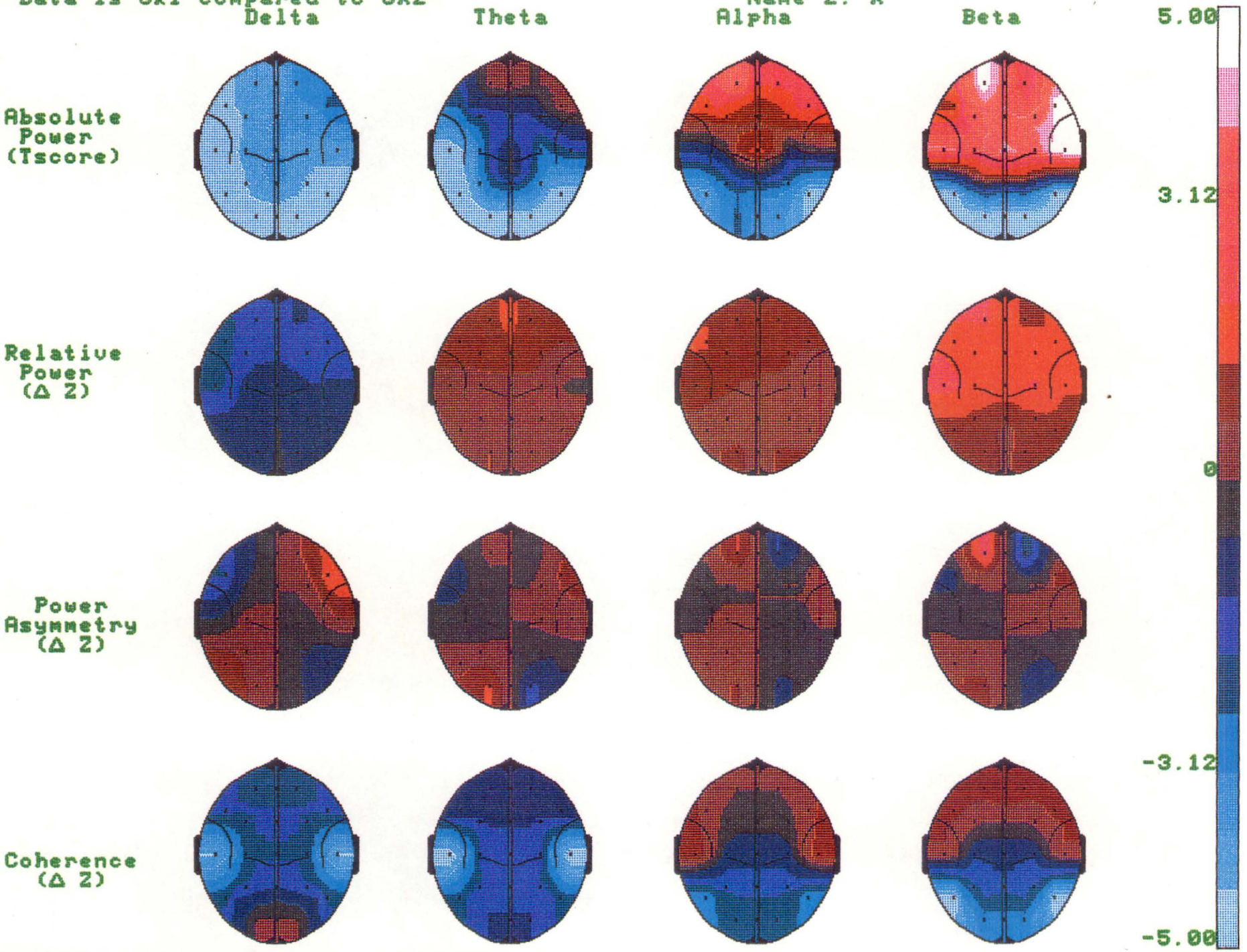
		Fp1	Fp2	F7	F8	F3	F4	C3	C4	Fpz	Fz	Cz
solute	Δ	-3.7	-2.9	-6.2	-2.5	-4.3	-3.4	-4.0	-4.0	-4.3	-3.4	-3.8
ower	θ	0.3	-0.0	-2.4	0.3	-0.9	-0.3	-3.1	-2.9	0.4	-2.5	-0.5
score)	α	3.5	2.1	1.7	2.1	1.5	2.2	-0.2	-0.9	1.9	-0.4	1.6
	β	5.7	1.9	1.1	4.8	2.5	3.6	2.8	3.3	3.1	3.2	3.9
lative	Δ	-1.5	-1.2	-2.1	-1.3	-1.4	-1.5	-0.9	-1.2	-1.5	-1.4	-0.8
ower	θ	1.1	0.9	1.3	0.6	1.1	1.0	0.5	0.5	1.5	1.1	0.5
Δ Z)	α	1.0	0.7	1.4	0.7	1.0	1.0	0.8	0.6	0.9	0.9	0.6
	β	2.0	1.2	1.8	1.4	1.6	1.6	1.8	2.1	1.5	2.0	1.7

ower	Δ	-0.5	-1.9	-0.4	0.4
mmetry	θ	0.3	-0.9	-0.5	-0.1
Δ Z)	α	1.3	-0.2	-0.5	0.5
	β	2.3	-0.8	-0.4	-0.4
erence	Δ	-2.0	-1.7	-1.6	-1.9
Δ Z)	θ	-1.0	-0.6	-1.1	-1.6
	α	0.0	0.6	-0.1	-1.0
	β	0.9	0.8	0.4	-1.1

		T3	T4	T5	T6	P3	P4	O1	O2	Pz	Oz
solute	Δ	-6.2	-4.0	-6.4	-8.1	-4.5	-5.0	-8.3	-7.6	-3.0	-7.0
ower	θ	-5.3	-1.8	-5.9	-7.6	-3.3	-4.0	-6.5	-8.3	-0.3	-6.4
score)	α	-1.1	-0.1	-4.3	-5.3	-2.6	-3.1	-2.3	-3.5	-2.4	-3.5
	β	3.3	5.3	-6.6	-8.7	-0.2	-1.4	-6.4	-7.6	-0.6	-8.1
lative	Δ	-2.1	-1.2	-0.7	-1.1	-0.6	-0.8	-1.0	-0.8	-0.6	-0.5
ower	θ	0.3	-0.1	0.5	0.6	0.4	0.6	0.7	0.3	0.7	0.5
Δ Z)	α	1.0	0.2	0.4	0.6	0.3	0.3	0.9	0.7	-0.0	0.2
	β	2.2	1.4	0.9	1.1	1.1	1.2	1.2	1.0	0.6	0.4

ower	Δ	-0.6	1.3	0.9	0.5
mmetry	θ	-0.5	0.5	0.2	1.5
Δ Z)	α	-0.1	0.4	0.3	0.7
	β	-0.2	0.6	0.6	0.8
herence	Δ	-4.5	-2.1	-1.3	0.0
Δ Z)	θ	-5.2	-2.3	-1.2	-1.2
	α	1.0	-3.1	-1.7	-2.0
	β	0.6	-4.9	-1.3	-2.8

Monopolar Correlated Tscore/Diff. Zscore Maps Name 1: GK1 F
Data is Gk1 compared to GK2 Name 2: K



Name 1: GK1 F
a is GK1 compared to GK2 Name 2: K
olar Correlated Tscore/Diff. Zscore Measures Analyzed: 06/12/90
rees of freedom: 37

		Central		Temporal		Parietal Occipital		Frontal Temporal	
		C3/Cz	C4/Cz	T3/T5	T4/T6	P3/O1	P4/O2	F7/T3	F8/T4
solute	Δ	-0.7	0.2	-0.2	1.6	-6.3	3.7	0.6	1.3
ower	θ	1.6	2.1	0.9	3.0	2.0	5.2	0.3	2.0
score)	α	2.7	2.4	-2.3	-0.3	1.9	2.4	0.7	0.4
	β	6.9	6.0	3.8	5.2	6.4	5.0	0.1	4.6
lative	Δ	-0.8	-0.8	-0.3	-0.3	-1.7	0.0	0.0	-0.2
ower	θ	0.1	-0.0	-0.1	0.0	1.2	0.1	-0.1	-0.3
Δ Z)	α	0.5	0.3	-0.5	-0.6	1.3	0.1	0.1	-0.3
	β	1.0	1.5	0.7	0.6	1.6	-0.3	-0.0	0.4

ower	Δ	-0.5	-0.7	-3.8	-0.5
mmetry	θ	-0.1	-0.8	-1.4	-0.7
Δ Z)	α	0.1	-0.5	-0.2	-0.1
	β	-1.1	-0.5	0.4	-0.8
erence	Δ	-0.4	-0.1	3.6	0.4
Δ Z)	θ	1.6	0.1	2.6	-0.3
	α	0.1	-0.6	1.8	0.5
	β	-0.1	-2.1	0.1	0.5

Bipolar Correlated Tscore/Diff. Zscore Maps
Data is Gk1 compared to GK2

Name 1: GK1 F
Name 2: K